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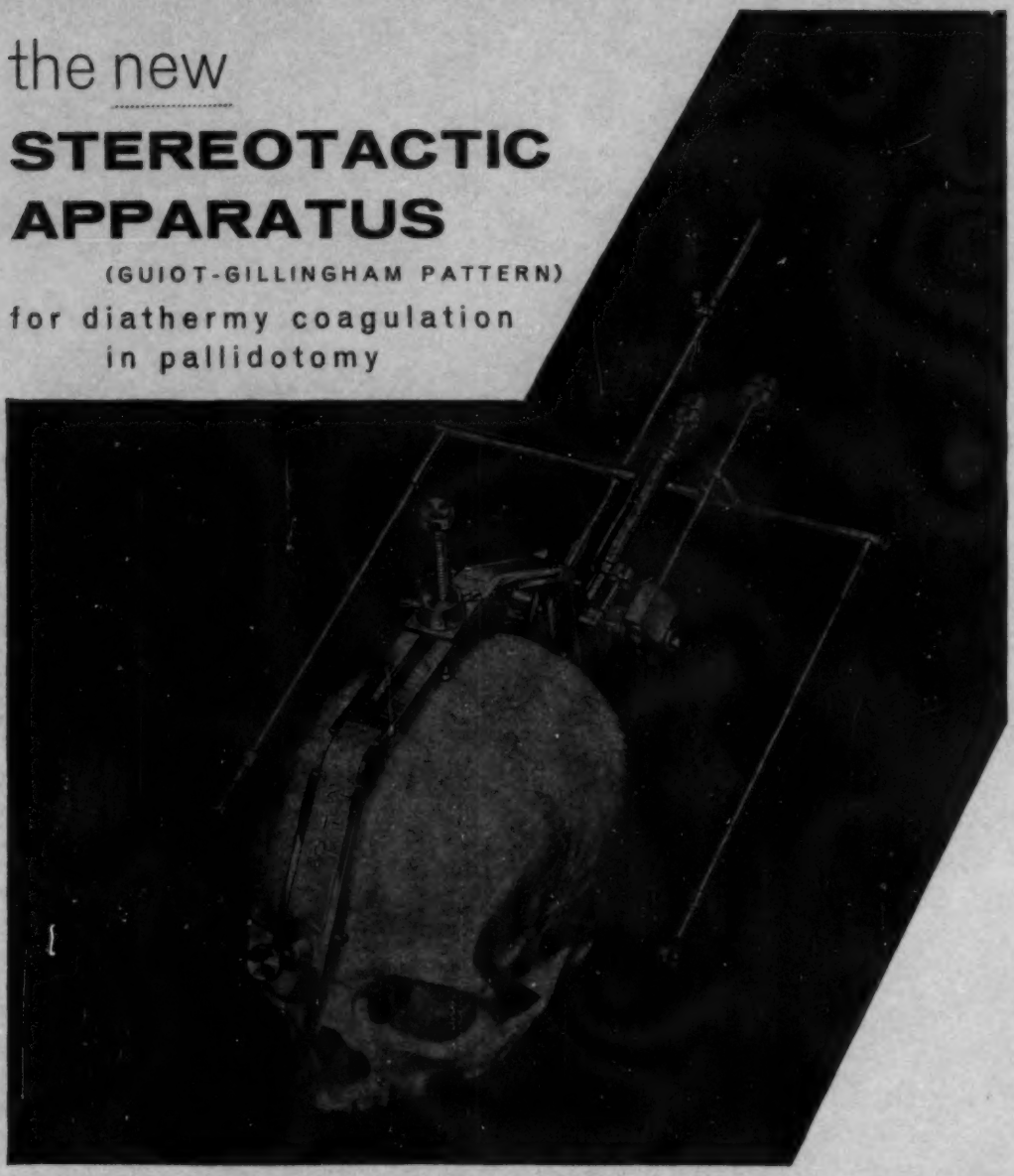

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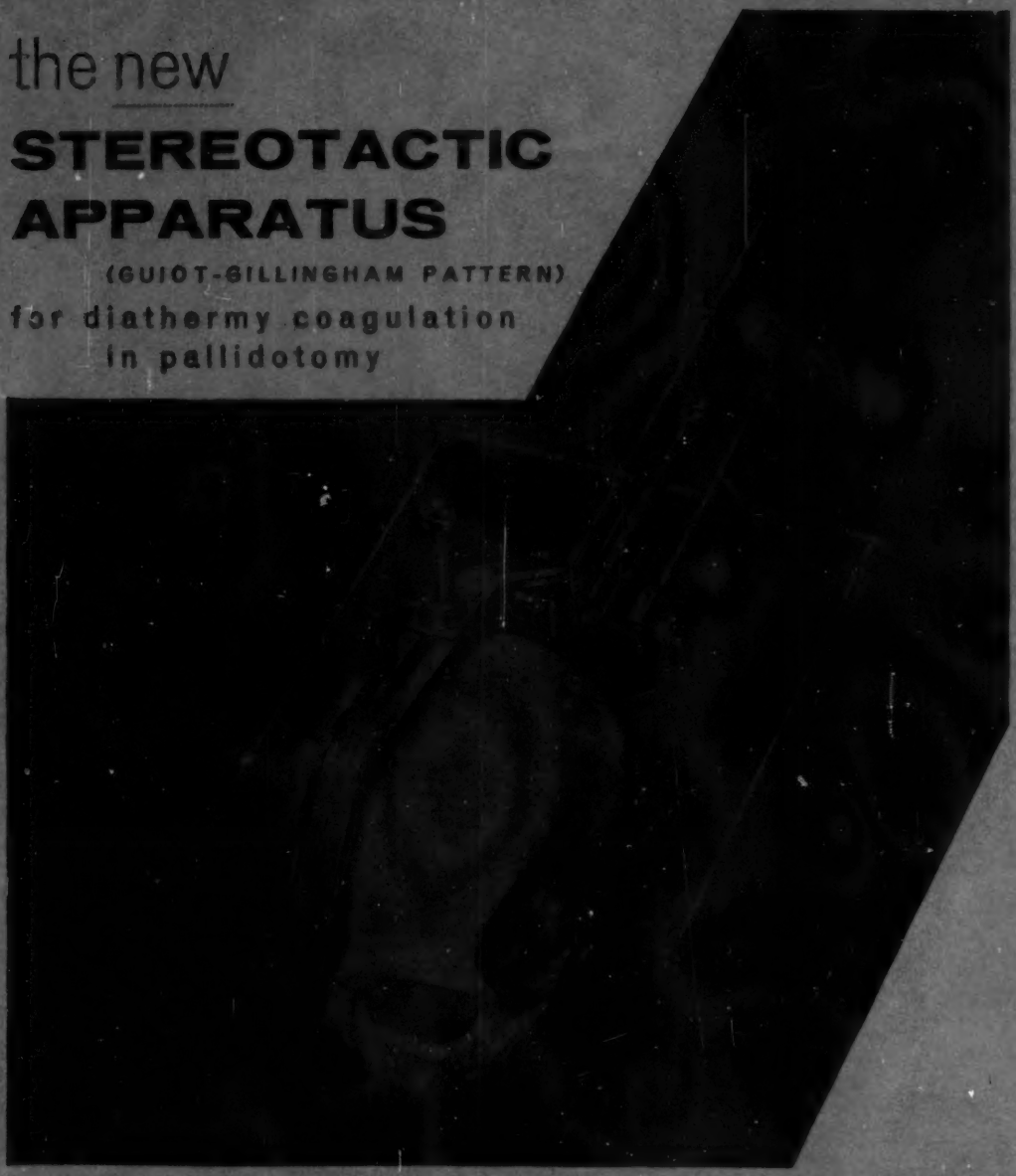

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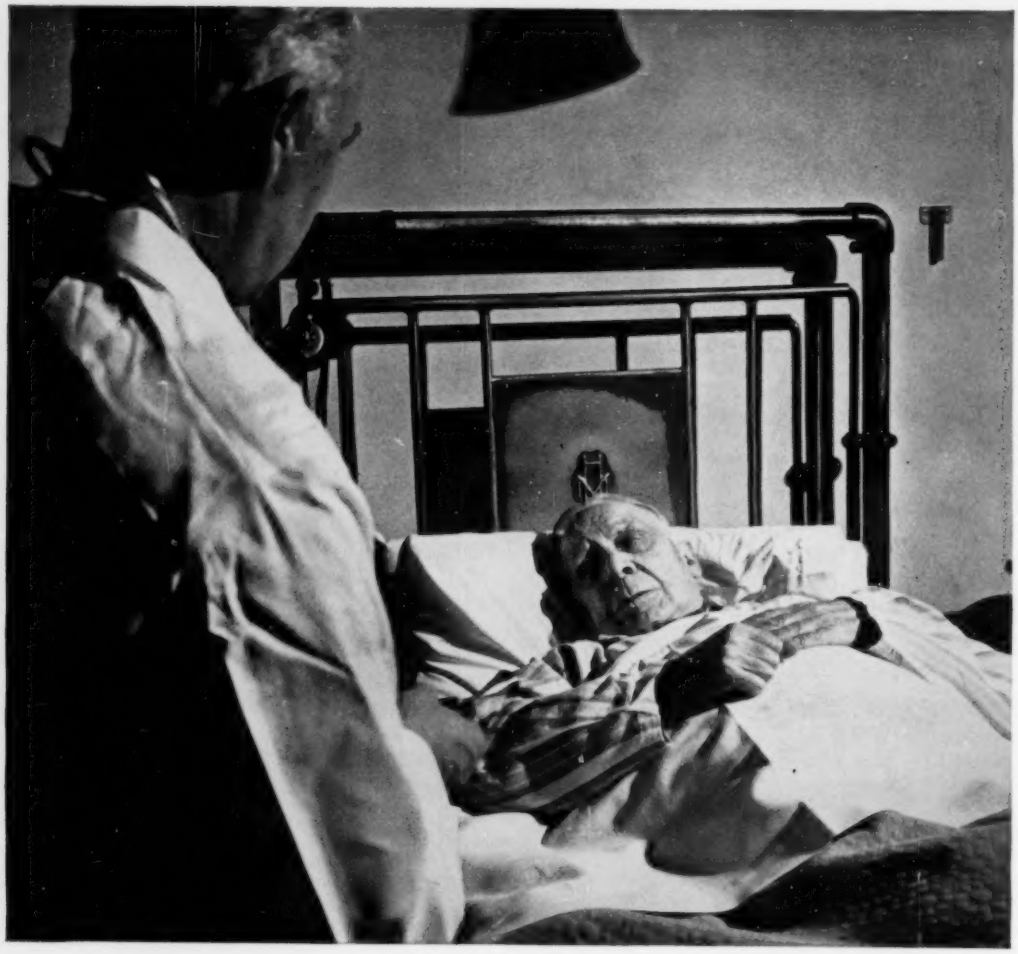

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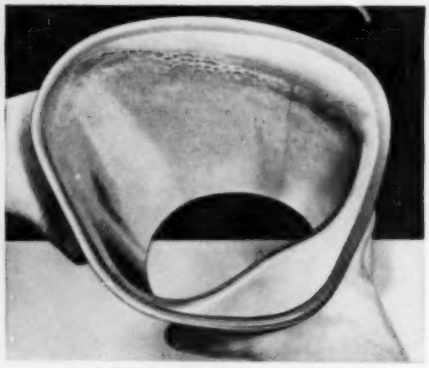

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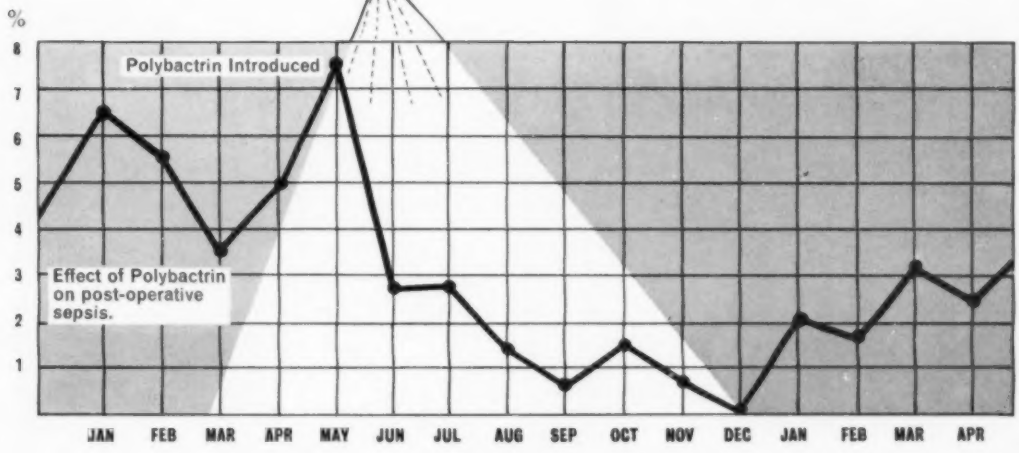

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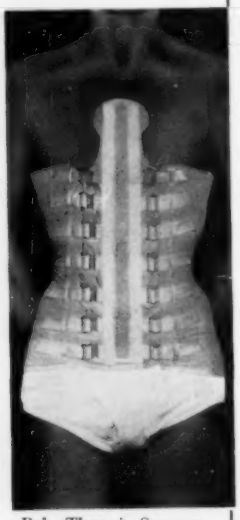

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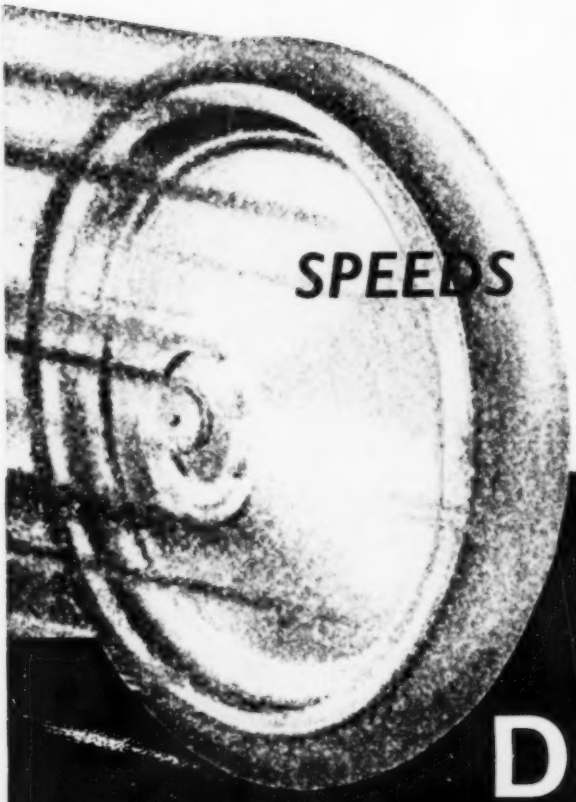

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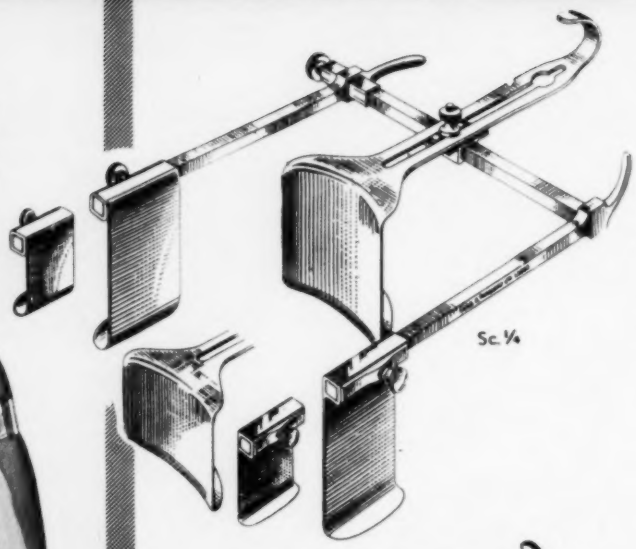

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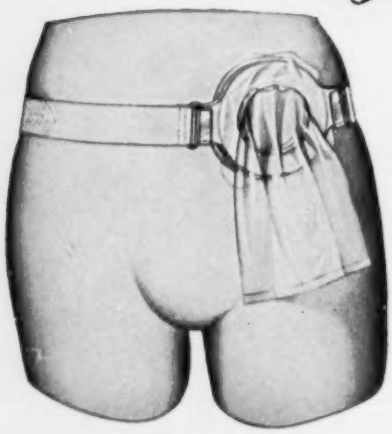

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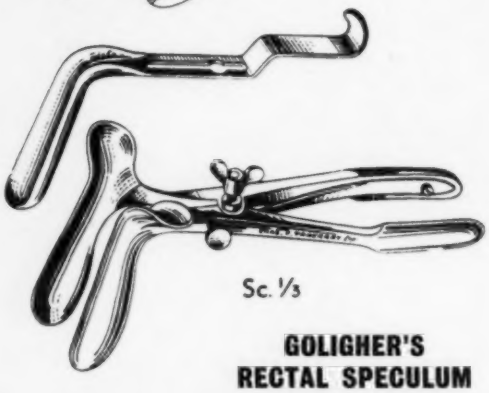

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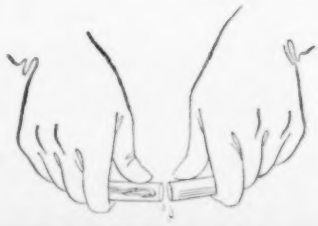

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CARCINOMA OF SALIVARY GLANDS SHOWING THE CYLINDROMA PATTERN

BY J. STEWART

FROM THE AYRSHIRE AREA LABORATORY, BALLOCHMYLE HOSPITAL, MAUCHLINE, AYRSHIRE

INTRODUCTION

AMONGST the tumours of salivary glands and of the mucous glands of the anterior foregut region there is recognized a group of neoplasms showing a characteristic histological appearance and variously referred to as 'cylindroma', 'basalioma', 'adenocystic basaloid carcinoma', and even other aliases. Billroth (1859) first used the expression 'cylindroma' to distinguish a tumour which having arisen in the nasal sinuses had invaded the orbit. The term was apparently a histological division without reference to prognosis and was inspired by the characteristic occurrence of columns of cells within a densely hyalinized 'cylinder' of collagen. It was many years before this lesion was to be identified as an entity of characteristic behaviour and prognostic significance. The history of this identification is well reviewed by Russell (1955), and a brief reference will be made to only some of the later literature on this subject. Dockerty and Mayo (1942), reviewing so-called 'mixed tumours' of the submaxillary gland, showed that those with the cylindroma pattern were widely infiltrative with selective invasion of nerves, high recurrence rate, and consequent poor prognosis. Mulligan (1943), reviewing 21 recorded cases of metastasizing mixed tumour, noted that in 8 the term 'cylindroma' was employed in the description of the histology, and in 4 more cases this appearance was present to judge from the descriptions given although not specifically stated. He suggested that the cylindroma pattern was correlated with the tendency to metastasize. Finally, in 1946, Quattlebaum, Dockerty, and Mayo separated the cylindroma from the mixed salivary tumour as a distinct entity for which they proposed adopting the term 'adenoid cystic carcinoma'. Despite this many texts still fail to distinguish these two groups of neoplasms, e.g., Willis (1955a), and it is easy to understand why this lesion, with its necessity for wide excision if treatment is to be successful, may sometimes escape recognition by surgeons and pathologists. It was therefore thought worth while to record the details of this neoplasm in 6 patients seen during the past five years and, using the material, to illustrate its characteristic histological features.

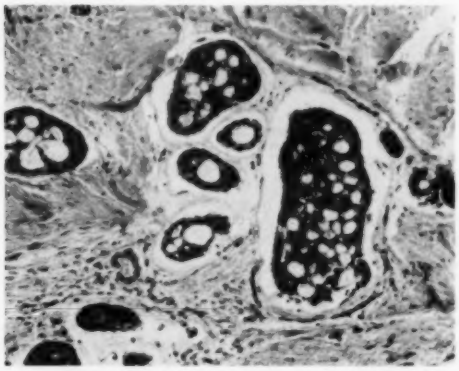

FIG. 270.—Showing the typical pattern of alveolar masses with variable cyst formation producing a cribriform pattern. H. and E. (× 50.)

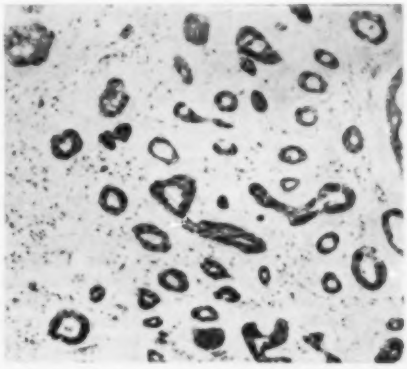

FIG. 271.—Section from the advancing edge of the neoplasm showing the characteristic columns or cylinders of cells to which the tumour owes its name of 'cylindroma'. H. and E. (× 50.)

SITE OF OCCURRENCE

These tumours occur in the mouth, nasal passages, pharynx, larynx, trachea, and bronchi, as well as in

the major salivary glands, and even occasionally in the lachrymal gland (Ranger, Lucas, and Thackray, 1956). In the major salivary glands they constitute only a small proportion of the tumours of this site, the benign mixed salivary adenoma being more frequent; but in the other sites the reverse obtains. In the larger air-passages the lesion is classified as a bronchial adenoma and usually qualified as 'cylindromatous variant'. Consequently, its identity with the neoplasm as it occurs elsewhere, in the nasopharynx, etc., may not be appreciated. *Case* 1 of this series, which presented initially as a lesion of the external auditory meatus, has been previously recorded by Johnstone, Lennox, and Watson (1957), who advanced the thesis that it was a variant of hidradenoma and identified it with these tumours as they occur elsewhere in the skin. For reasons which are advanced later it is suggested that the external auditory meatus may be a primary site of occurrence of cylindroma as an ectopic neoplasm identical with those of the anterior foregut.

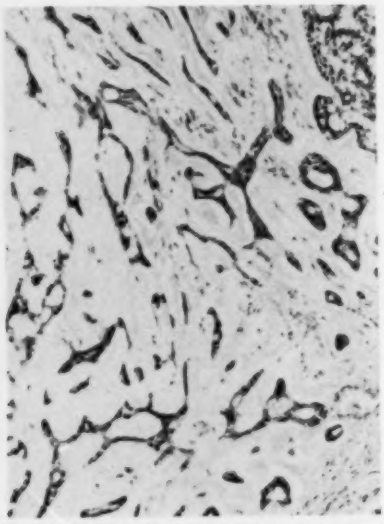

FIG. 272.—This view is from a densely hyalinized tumour in which the neoplastic cells form a reticular pattern or coarser cribriform pattern. H. and E. (× 50.)

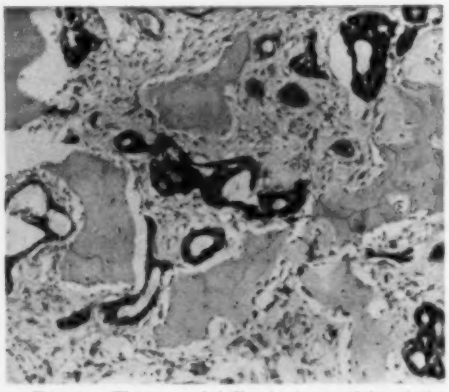

FIG. 273.—The tumour is infiltrating bone and the spicules of cancellous bone are quite unaltered but there is some reactive fibrosis of the medulla. H. and E. (× 50.)

HISTOLOGY

To avoid unnecessary repetition the microscopical features of each case will not be described in detail. Typically, the tumour occurs in alveolar masses of small cells with darkly staining nuclei and scant cytoplasm with poorly defined cell boundaries. Mitotic figures are rare. Within these masses cysts of varying size occur producing a cribriform pattern (*Fig.* 270). It is at the periphery or spreading edge, the youngest part of the tumour, that the cylindromatous pattern is best seen. This derives from the incidence of cells in columns with centrally situated cystic space (*Fig.* 271). The tumour cells spread within connective-tissue planes rather than directly into the tissues in the aggressive fashion of a frank carcinoma, so that the tumour is amongst the tissue but not really of it. This curious relationship to the surrounding tissues is marked by a narrow interval zone which may be unstained in the sections or show progressive eosinophilia with its conversion to hyaline tissue. The cysts within the cell masses are in many instances in continuity with this narrow boundary zone and show the same staining affinities. These cystic spaces probably derive from cell division occurring in restricted planes, so that the tumour comes to form a spongework of cells. The nature of these cysts and the interval zone is of some interest. Many authors report positive mucin staining in these areas, but more specific techniques (Azzopardi and Smith, 1959) have demonstrated this to be of connective-tissue type. The tumour masses are bathed in this apparent connective-tissue ground substance which subsequently is transformed to hyaline tissue. This appears to be a reaction of the host tissues rather than a secretion of the neoplastic cells. With progressive hyalinization the tumour is usually of a coarser cribriform or even reticular pattern (*Fig.* 272), and at this stage the lesion is present as a firm or hard mass. Epithelial mucin is also demonstrable, but is much less abundant in its incidence and occurs within small ducts which are quite easily discernible. They are lined by a larger cell with well-defined eosinophilic cytoplasm and pale vesicular nucleus.

The striking infiltrating habit of growth is well demonstrated where bone is encountered. *Fig.* 273 illustrates the quiet occurrence of the lesion within the bone, where it has reached via the Haversian systems with striking absence of reaction and no resorption of bone, which remains functionally intact. Perineural infiltration (*Fig.* 274) is almost a classical feature which is invariably illustrated in articles on this neoplasm and it probably explains the pain which is frequently prominent in the symptomatology.

Whilst the above is a description of this lesion in its most typical pattern, occasionally, as in 2 of our cases, the cellular components of these lesions are larger with vesicular nuclei showing a single prominent nucleolus. It is in this type that necrosis of the cell masses is seen (*Fig.* 275), and with more

aggression in its infiltrating habit suggests a relatively more malignant variant. *Case* 2 and *Case* 5 of this series, which showed this large cell pattern, were both to be dead within two years of presentation. Patey and Thackray (1958) draw attention to this added danger of these tumours undergoing frank malignant change with enhanced aggression both locally and in their metastases, which are more frequent and extensive.

CASE REPORTS

Case 1.—Male aged 53 years. The patient was first operated on in 1948 for a tumour in the wall of the external auditory meatus known to be there during the past two years and causing episodic pain. In a series of recurrences it was variously reported as 'basal-cell carcinoma' and 'epithelioma adenoides cysticum' before finally it was recognized as a 'cylindroma'. This patient has averaged more than one recurrence each year. It is interesting to note that these tend to occur in a somewhat radial distribution from the primary presentation in the auditory meatus, and at his last visit in 1958 the facial nerve was infiltrated, for which reason it was not excised at this time. In February, 1958, two small pulmonary metastases were recorded. Review to date shows gradual increase in size and number of lung lesions.

Case 2.—Male aged 53 years. The patient first attended the E.N.T. clinic in May, 1954, complaining of recent onset of pain in a swelling of the roof of the mouth and right cheek present during the past five years. A slight swelling, tender to pressure, was observed at the right side of the palate and X-ray examination showed destruction of the medial wall of the right antrum. A Caldwell-Luc antrostomy was performed and several biopsies taken at this time all showed a cribriform cylindroma. The lesion was widely excised and a prosthesis fitted. He remained well for one year, when he returned with

FIG. 274.—The lesion is mantling a large nerve-trunk. H. and E. (× 50.)

recurrent neoplasm causing proptosis of the right eye. This was enucleated and the orbital cavity cleared. A month later he was noted to have metastasis in the cervical vertebræ and died a year later at home. Autopsy was not performed. Microscopically the neoplasm in this case was of the larger cell type.

Case 3.—Female aged 37 years. The patient was first seen at the E.N.T. clinic in 1954, complaining of increasing nasal obstruction during the past year. There had been occasional epistaxis during the previous three months and purulent discharge was now present. A biopsy of a posterior pharyngeal tumour showed 'cylindroma', and this was subsequently excised as widely as possible. Histological examination showed excision to be incomplete, but surgical intervention was not again necessary until 1958, when it was widely cauterized by diathermy. Chest radiography in 1957 for the purposes of her employment revealed multiple small nodular (?) metastases. Review to date shows that these remain apparently quiescent.

Case 4.—Male aged 73 years. The patient was referred in March, 1956, to the E.N.T. clinic for investigation of recurrent epistaxis. Radiography showed a loss of translucency of the left antrum, with destruction of the lateral wall. At operation extensive infiltration of the wall

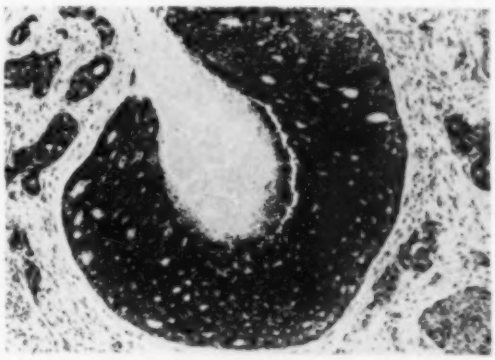

FIG. 275.—This is a typical pattern of the malignant variant of this tumour. The cells are larger and the cysts less prominent. Depending upon the plane of section the area of necrosis may appear to be central, but its true relationship is well seen here. H. and E. (× 93.)

of the antrum, which was filled with tumour, was found. A biopsy was taken and he was subsequently treated by radiotherapy. Initial response to this was satisfactory, but recurrence is now closing the nasopharynx and repeated epistaxis is again troublesome. The patient's general condition does not permit of any further active treatment.

Case 5.—Male aged 70 years. The patient was first seen when complaining of a swelling over the right maxilla which had increased in size during the previous two months. No pain was complained of, but X-ray examination showed destruction of the maxilla and right lateral antral wall. Biopsy showed a 'cylindroma', and the lesion was widely excised. He remained well until eighteen months later, when he was considered to be suffering from intracranial invasion. He was then complaining of headache, 'black-outs', increasing deafness, and failing vision. The patient died at home but unfortunately an autopsy was not permitted to confirm invasion of the skull. This lesion was histologically similar to *Case* 2, i.e., larger cell type.

Case 6.—Female aged 56 years. The patient first noticed a swelling under her right jaw two months before admission. She admitted to occasional pain shooting up the right side of the face. After biopsy had shown the lesion to be a 'cylindroma' sited in the floor of the mouth, wide excision was carried out. At operation the lesion was extending into the base of the tongue, anterolateral aspects of the pharynx, and the tonsils of the right side. It was necessary to sacrifice a large portion of the tongue in the attempted ablation and the patient subsequently developed bronchopneumonia from which she died. A post-mortem examination was not permitted.

DISCUSSION

The incidence of this neoplasm in the anterior foregut, including the major salivary glands and respiratory tract, i.e., the site of occurrence of mucous glands, is strong argument for assuming a relationship

between the mucous glands and the neoplasm. Indeed, the incidence in major salivary glands has for many years caused it to be confused with benign mixed salivary tumour. The occurrence of ducts containing epithelial mucin has prompted statements that the lesion takes its origin from the duct epithelium of the mucous glands. The incidence of ducts in late recurrences of *Case* 1 indicates that this is an expression of differentiation within the tumour.

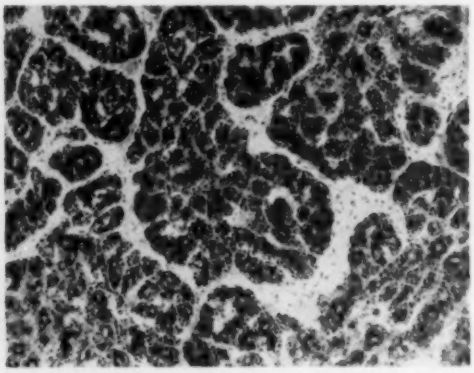

FIG. 276.—This well-differentiated lesion with ducts and acini organized in a lobular pattern was seen in association with typical adenocystic basaloid carcinoma. H. and E. (× 50.)

Even more striking differentiation was present in *Case* 4. *Fig.* 276 illustrates the lobular incidence similar to that seen in the normal gland and many ducts are present, but the bulk of the tumour is apparently an abortive acinar-cell anlage. The material from this case was fragmentary, but the occurrence of areas showing the cribriform and cylindromatous pattern leaves no doubt about the diagnosis.

The behaviour of these tumours is aptly summarized as "relentlessly recurrent" (Pollack, 1952). This is a result of several factors such as the late stage at which these lesions declare themselves by symptoms and the infiltrating habit of growth which means that the lesion is usually widespread when it reaches the surgeon. Finally, there are the technical difficulties in dealing with the lesions in relatively inaccessible sites. Recognition of the lesion should, therefore, lead to a most energetic and radical attempt at excision with as wide a margin of normal tissue as possible. Unfortunately, it is in a very small proportion of cases that excision will be feasible and our experience with the last case of this series, which was *mort guérie*, has suggested that sometimes it is better to deal with the lesion symptomatically. For the latter, surgery or radiotherapy appear to be equally suitable (Ranger and others, 1956). The lesion is radio-sensitive, but results are as disappointing as surgery in dealing with the lesion either initially or in its recurrences. It may be that this is due to lack of understanding of the widely infiltrating habit of growth and consequent failure to treat a suitable wide area. The presence of distant metastasis which is most frequently in the lungs, as in two of the cases recorded, does not appear to indicate immediate worsening of the prognosis. In both our cases, one of which has been under observation for two years, there has been very little radiological evidence of progress, although it must be assumed that the lesions are slowly advancing.

In any discussion of this lesion it is necessary to say something about nomenclature. Some authors have suggested an affinity with basal-cell carcinoma of the skin and this has led to the alternative name of 'basalioma' for these lesions (Ringertz, 1938). 'Cylindroma' is inaccurate as some may not show this typical pattern and most show it only at the periphery of the lesions. It also suffers the demerit of conveying nothing of its grave prognostic significance to the surgeon unfamiliar with the entity. 'Adenoid cystic carcinoma', the term favoured by American authors, is sometimes criticized as it fails to suggest affinity with the basal-cell group. 'Adenocystic basaloid carcinoma' would seem to meet this criticism and is used by Anderson (1953).

A proportion of adenocystic basaloid carcinomas, especially when presenting in the nasal passages, are misdiagnosed as 'adamantinoma', and indeed are sometimes thought of as synonymous by pathologists as well as surgeons. Willis (1955b) must accept responsibility for some of this confusion by his energetic statement of the case for adamantinoma. If adenocystic basaloid carcinoma is not a common neoplasm, adamantinoma is decidedly rare. It is agreed that many sections from these lesions must be examined before an opinion is given, but it is to be doubted if the occurrence of a 'stellate reticulum' within the masses of cells is proof of adamantinoma.

In describing the site of occurrence of this neoplasm we mentioned the external auditory meatus and reference was made to a paper by Johnstone and others (1957). Two of the five cases in that paper showed the cylindroma pattern—*Case* 3 is our *Case* 1, which remains recurrent, and *Case* 4 was cured after a recurrence, excision of which included part of the parotid gland. The possibility of these lesions originating in parotid gland or ectopic mucous glands was not considered. The behaviour of this neoplasm is in such marked contrast with hidradenoma of the skin showing the cylindroma pattern that it suggests an identity with the anterior foregut neoplasm. The logical consequence of this is to recommend that such tumours of the external ear should be treated by excision, including at least an appropriate portion of the parotid gland. Even some reported basal-cell carcinomas of skin of the cheek over the parotid which remain recurrent after wide excision may be missed examples of adenocystic basaloid carcinoma of the underlying salivary gland (Rank and Wakefield, 1958). Incidentally, in their excellent article, although dealing with the surgery of basal-cell carcinoma, these authors have many things to say about treatment that apply equally to adenocystic basaloid carcinoma.

It is obvious from the literature on this subject that the same lesion at different sites enjoys a different name, and sometimes even several aliases are available for the same site. This custom is to be deplored as an unnecessary source of confusion and the same name should be used for this entity whether in a salivary gland, the nasopharynx, the oropharynx, the trachea, or the bronchi. Our preference is for 'adenocystic basaloid carcinoma'. It is admittedly an omnibus

expression, but carcinoma should be included to indicate the malignant nature of the growth, whilst this is qualified by 'basaloid', which is useful in two respects—to qualify carcinoma to which exception may otherwise be taken, and to suggest an affinity with the basal-cell growths of the skin. 'Adenocystic' is a morphological expression and in our experience more appropriate than cylindroma.

SUMMARY

Six cases of mucous gland tumour of cylindroma pattern occurring in the foregut region are briefly reviewed and the occasion taken to illustrate the typical histological features of this growth. The site of occurrence is discussed and it is suggested that the external auditory meatus may be an additional site of presentation, if not indeed, of origin. The confusion of nomenclature that tends to submerge this entity is reviewed, and from a wealth of aliases 'adenocystic basaloid carcinoma' is recommended to distinguish this lesion at whatever site it occurs.

My thanks are due to Dr. J. A. Milne, for his advice and encouragement, and to Mr. J. Robertson, F.I.M.L.T., for technical assistance.

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A PUMP-OXYGENATOR FOR WHOLE-BODY AND REGIONAL PERFUSION

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IN extracorporeal units, the pump circuit can be arranged so as to provide either an output which is regulated by the venous return or one which is independent of it: experience with whole-body and regional perfusion in this laboratory suggested that the latter might be preferable.

In this paper an extracorporeal unit designed and built at this hospital and capable of maintaining a pump output independent of the venous return is described and some of its features are briefly discussed. At the time of writing this unit has been used successfully for whole-body perfusion in the laboratory and clinically for the perfusion of isolated vascular beds in the treatment of malignant disease.

THE EXTRACORPOREAL UNIT

The pump employed was the Ormonde-Mono pump (Hall, James, Lucas, and Waterston, 1959) driven by the Kopp Size 14 Type MS ½ h.p. Buxton certified flashproof motor and continuously variable gear. Since the unit was designed for the perfusion of isolated vascular beds as well as the whole body, a range of outputs from 70 ml. to 5000 ml. was required and this was provided by placing a belt drive with interchangeable cogs and a fixed 1 : 2 reduction between the variable gear and the pump so that by selecting the appropriate pair of cogs and stator (a component of the pump) one of the following ranges of output could be selected over which the pump output would be continuously variable during perfusion: 70 ml./min.–625 ml./min., 145 ml./min.–1255 ml./min., 290 ml./min.–2500 ml./min., 320 ml./min.–2810 ml./min., and 640 ml./min.–5620 ml./min. Provision for mechanical or electrical failure was made by a manual control which disconnected the pump from the final drive and connected it to a handle by which the pump could be turned by hand. The motor, gears, and pump were connected to a rigid baseplate and this in turn was connected to the chassis by shock-absorbing bearings to reduce noise. The chassis, which carried a drip-stand, was mounted on wheels and could be locked to the floor by bolts placed at its corners. These features are illustrated in *Fig.* 277.

The output of the Mono pump is a linear function of the speed at which the pump is turning, and since the pump (with the rubber stator) is very nearly occlusive, this output is for practical purposes independent of the range of resistances encountered in perfusion work. A revolution counter with a scale calibrated in ml. per min. could therefore be placed on the pump shaft and then functioned as an arterial flowmeter accurate to ±5 per cent. An additional means of measuring the pump output was available since the reduction given by the variable gear was shown numerically on a counter attached to the gear control (*see Fig.* 227). The numbers shown on this counter were related to the pump output by prior calibration and the gear control then monitored as well as regulated the pump output.

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The hæmolysis rate of this pump is acceptably low (Hall, James, Lucas, and Waterston, 1958).

To make the pump independent of fluctuations in the venous return a reservoir was placed between the pump and the oxygenator. This incorporated a disposable stainless-steel mesh filter and was so designed as to function as a bubble-trap (*Fig.* 278). Since the volume of blood in the oxygenator used was constant at any given flow-rate the reservoir was the only part of the circuit whose volume was variable and, by calibrating it, alterations in the volume of blood in the extracorporeal unit could be monitored continuously by reference to the blood-level in the reservoir. In a reservoir with a maximum capacity of 3000 ml., 50 ml. shifts were easily detected (*see Fig.* 278) and, for the perfusion of isolated vascular beds or for whole-body perfusions in small patients or dogs, slimmer reservoirs were available with a smaller capacity and a sensitivity down to 10 ml.

The dangers of a circuit in which the pump is independent of the venous return and draws directly upon a reservoir are twofold: (1) If the venous return should suddenly become very much reduced, e.g., by hæmorrhage, the reservoir may empty so that the pump pumps air; and (2) If the venous lines become obstructed and the pump output remains unaltered, the volume of blood in the circulation being perfused may become dangerously increased. To guard against these dangers a photo-electric device was incorporated to switch off the pump when the reservoir level fell below a preselected point. This device could be over-ridden by the pump operator and the pump was then governed by an on/off switch on the control panel.

A danger that emerged with the industrial version of the Mono pump was that the pump could not reliably be freed of air when first primed so that bubbles were sometimes released during perfusion. This danger has been eliminated in the Ormonde-Mono pump (Hall, James, Lucas, Waterston, 1959). Nevertheless, the circuit was so arranged that the saline circulated before priming the unit with blood could be drained by gravity from the whole of the circuit except the pump through a nipple at the bottom of the reservoir. The fact that the pump, being below the bottom of the reservoir, could not be emptied of saline with the rest of the circuit ensured that no air entered the pump when the saline was drained and replaced with blood.

It is theoretically undesirable to subject blood in an extracorporeal unit to subatmospheric pressures since this raises the possibility of gases coming out of solution to form discrete bubbles. Such pressures may be generated in the afferent line to the pump. In the circuit described, however, the blood in the afferent line was under a positive hydrostatic pressure since the pump was beneath the reservoir. This pressure was reduced by the sucking action of the pump, but the internal diameters of the afferent line and of the nipples to which it was connected were selected to ensure that the afferent-line pressure did

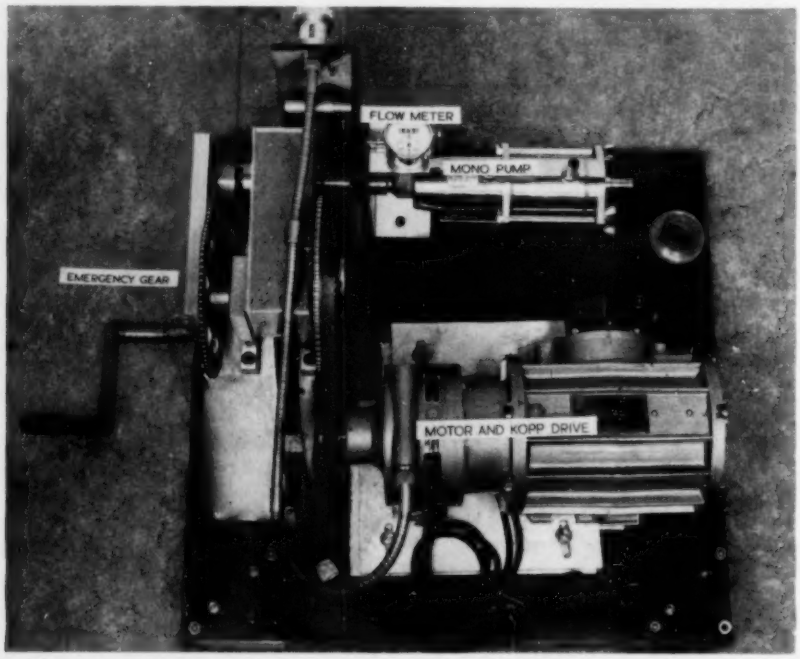

FIG. 277.—The pump components revealed by removing the top of the chassis. The revolution counter has not been recalibrated and connected. The socket for the drip-stand can be seen to the right of the pump.

not become subatmospheric at flow-rates up to 20 litres per min.

A removable cover was provided for the motor and gears and the top of this cover was left clear so that a heat exchanger and or oxygenator could be placed upon it. We have used the Aga oxygenator which is shown mounted on the chassis in *Fig.* 279.

Blood flowed under gravity from the oxygenator into the reservoir and the possibility therefore arose that bubbles might be introduced into the blood-stream if there was a free fall to the blood-level in the outflow tube from the oxygenator of more than 1·5 cm. (Senning, 1959). To eliminate this danger the reservoir was placed on a vertically adjustable stand (*see Fig.* 279) so that by altering the distance between the reservoir and the oxygenator the level in the outflow tube from the oxygenator could be kept up, in spite of alterations in the volume of blood in the reservoir. In addition, the photo-electric cut-out was set to switch the pump off if the level in the outflow tube fell 1·5 cm. and during perfusion the outflow end of the oxygenator was kept flooded with a measured volume of blood.

Venous return was by gravity drainage: the top of the chassis was 13 in. above the floor and this distance could be increased to 14·4 in. by jacking up the chassis with the bolts at its corners. With the Aga oxygenator the blood fell to a point 22–23·5 in. above the floor. A clamp on the venous line controlled from the control panel allowed the venous return to be progressively reduced when necessary. Ports were provided at the venous end of the oxygenator for the return of blood from the sucker and for the connexion of standard giving sets through which blood or saline could be introduced into the circuit.

The priming volume of the extracorporeal unit varied with the oxygenating capacity required and with the initial volume in the reservoir. The latter depended upon the expected flow-rate since the efficiency of the reservoir as a bubble trap varied inversely with the flow-rate and directly with the volume of blood contained in it and since the greater the flow-rate the more blood was required in the reservoir in order to keep the emptying-time of the reservoir acceptably high. Using one Aga oxygenator tray, 1½ litres primed the circuit and left approximately 200 ml. in the reservoir.

The components which came into contact with blood and the tubing connecting them could be removed from the chassis without breaking the tubing circuit (*Fig.* 280) and hence these components could be assembled for use in a non-sterile fashion, tested, sterilized, and then placed on the chassis. Thus once sterilized the circuit was never opened and no assembly of sterile components was necessary. Sterilization was achieved at room temperature by exposure to ethylene oxide gas (Freeman and Barwell, 1960) which penetrated the tubing by solution in the plastic and the oxygenator via the gas ports.

DISCUSSION

A pumping arrangement in which the output is immediately under the control of the pump operator and in which the pump draws directly upon a reservoir enables the output to be kept constant or readily altered independently of the venous return. The maintenance of a constant output seems desirable in the face of hæmorrhage and expansion of the vascular bed, both of which cause falls in the venous return, while some evidence was obtained in this laboratory which suggested that a fluctuating output during perfusion was deleterious in itself. Alteration of the flow-rate during regional or whole-body perfusion is occasionally necessary—for instance, if the calculated flow-rate proves inappropriate once perfusion is established—and under these circumstances it is an advantage if the pump output can be easily and accurately adjusted.

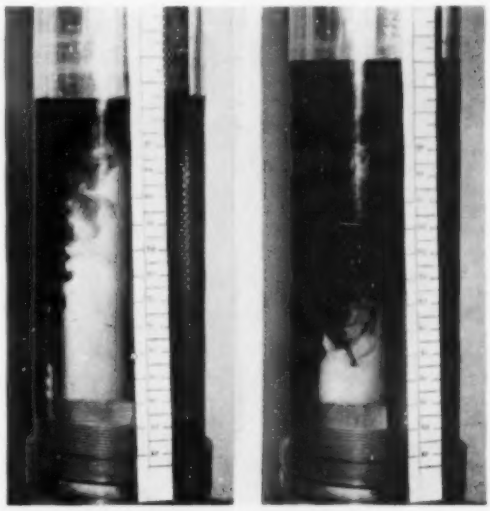

FIG. 278.—The streaming of fluid within the reservoir upon which the latter's action as a bubble trap depends. In this figure water is being recirculated through the extracorporeal unit and a dye has been injected into the oxygenator so as to colour the fluid reaching the reservoir. The inflow and outflow nipples are placed at opposite sides of the base of the reservoir and the inflow nipple directs the blood vertically upwards. The inflow nipple is of a smaller bore than the outflow nipple and, to reach the latter, blood must pass through a vertical cylindrical filter of fine stainless-steel mesh. (A coarse filter is shown in this photograph.)

A, Illustrating the absence of mixing between the inflow and outflow streams below the surface. Bubbles entering the reservoir therefore tend to rise to the surface (aided by gravity) rather than to be drawn over to the outflow stream. As the flow-rate rises the drag from inflow to outflow streams increases, but this is balanced by the increased upward velocity of the incoming stream. B, Illustrating the state of affairs shortly after that shown in A. As can be seen, there is no true outflow stream in the upper part of the reservoir: an effect produced by the turbulence due to the filter and to the relatively wide bore of the outflow nipple. The efficiency of the reservoir as a bubble trap and the filtration area depend upon the quantity of blood in the reservoir. The minimum level at which this should be maintained depends upon the flow-rate.

The attraction of the alternative arrangement, in which the pump output is locked to the venous return, is that at all times during perfusion the circuit contains a constant volume of blood and, hence, provided that the total volume of blood circulating in both patient and machine remains unaltered, it may be assumed that the patient's blood-volume has also remained constant. A possible disadvantage of this arrangement, however, is illustrated by the finding of Donald and Moffitt (1956) that during perfusion of the dog using a machine of this type, a

withdrawal of blood amounting to 10 per cent of the blood-volume reduced the rate of flow by 50 per cent and that the flow continued at this reduced level until the volume abstracted was returned. In this laboratory it has been found difficult to add blood from the venous side of such a pump at a rate that exactly such times so that vascular filling and the blood-pressure tended to be maintained.

The effect of a fluctuating output during perfusion was suggested by the finding that 8 out of the first 10 dogs perfused at a constant flow-rate survived, whereas 3 dogs, subsequently perfused with the same

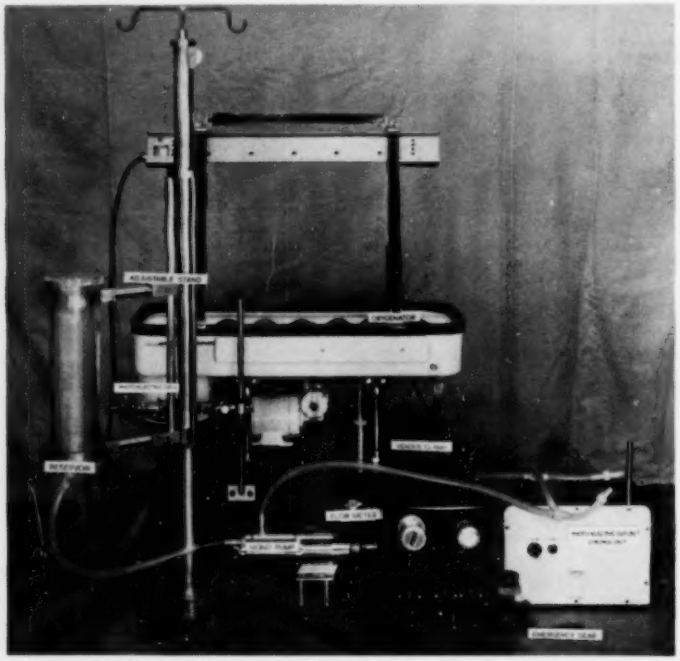

FIG. 279.—The pump operator's view of the extracorporeal unit. The controls, grouped on the right, are (from above down and left to right): the arterial output control, the venous return control, the pump on/off switch, the Aga oxygenator motor on/off switch, the photo-electric cut-out circuit on/off switch (this over-rides the photo-electric cut-out circuit), and the Aga oxygenator heaters on/off switches (half and full heat). The Aga oxygenator tray and its motor can be removed from the top of the chassis to leave this space clear. A second Aga oxygenator tray can be attached to the back of the chassis with removable brackets and can, if desired, be used alone, leaving the top of the chassis clear for a heat exchanger.

offsets falls in the venous return and hence such a pump tended to produce a fluctuating output.

In contrast, our experience during whole-body by-pass in the dog with the circuit described in this paper was similar to that of Gianelli (Gianelli, Robinson, Best, and Kirby, 1958) in that, by maintaining a constant pump output in the face of hæmorrhage, the blood lost was found to deplete the volume of blood in the extracorporeal unit (i.e., in the reservoir) and not the volume of blood in the dog. This was regarded as desirable since the animal's blood-volume and circulation rate were for practical purposes unaltered by hæmorrhage and so closely was the reservoir level affected by the latter that it was found possible to estimate accurately the volume of blood lost during by-pass by noting the fall in the volume of blood in the reservoir during this time. It was also noted that expansion of the vascular bed during perfusion resulted in a fall in the venous return and hence, if the pump output was kept constant, the animal's blood-volume was increased at pump and technique but at a deliberately fluctuating flow-rate, all died in the early post-operative period. Of the 2 non-survivors in the first series, 1 died 24 hours after by-pass with a hæmothorax and the second made a normal recovery until it was seen on the E.C.G. to suffer a cardiac arrest 7 hours after by-pass. The cause of death in the second series was not obvious at post-mortem, but immediately following by-pass all dogs developed a progressively falling arterial blood-pressure and venous oxygen saturation whilst the venous pressure and arterial oxygen saturation remained normal. It seemed possible from the data available that death in this series had been due to a falling cardiac output possibly secondary to a loss of peripheral vascular tone, and that this had been related to a period of whole-body by-pass during which the flow-rate had fluctuated.

It is sometimes necessary to alter the flow-rate during perfusion, and with a pump whose output is regulated by the venous return such adjustments can only be made by augmenting or depleting the venous

return. If gravity drainage rather than a venous pump is employed, this involves adjusting the amount of blood in a reservoir on the venous side of the circuit. In our experience, this method of altering the flow-rate is more cumbersome and less precise than the alternative of using an arterial reservoir and a pump, the output of which can be directly adjusted by the pump operator.

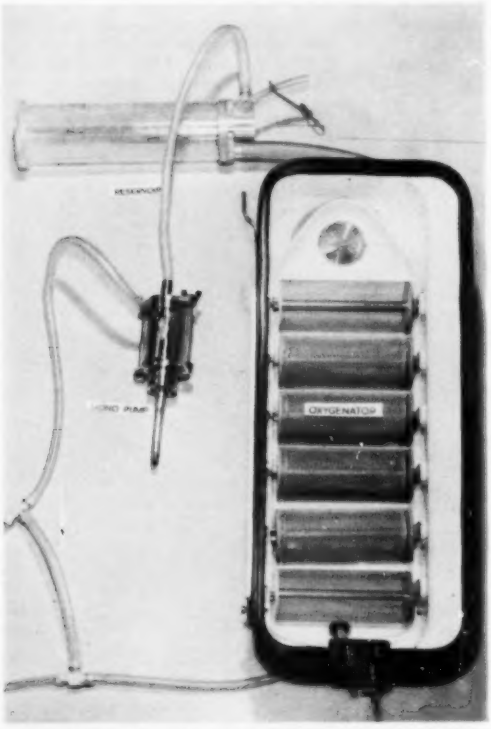

FIG. 280.—The extracorporeal circuit assembled and detached from the chassis ready for sterilizing.

Control of the venous return with a clamp allowed the venous pressure during perfusion to be regulated and the venous return to be progressively reduced after the caval slings had been loosened at the end of by-pass. By concurrent reduction of the pump output, the heart could then be presented with a gradually increasing proportion of the minute-volume rather than with the whole of the venous return at once. If necessary, adjustment of the venous return and pump output out of phase with each other allowed alterations to be made in the animal's blood-volume at this time.

For the perfusion of isolated vascular beds in the treatment of malignant disease a pump of the type described in this paper was preferred, since an initial adjustment of the pump output was often necessary because the blood-pressure generated by any given output was difficult to estimate in advance and since, once an appropriate output had been obtained, its constant maintenance allowed net leaks in and out of the 'isolated' circulation to be detected by noting changes in the reservoir level. (Although knowledge of the net leak is valuable, it is of course of greater practical importance to know the size of the leak out of the isolated circulation.)

Although not a feature peculiar to pump circuits of the type here described, the arrangement whereby the whole of the circuit requiring sterilization could be assembled on the chassis, tested, closed, removed intact, sterilized, and replaced, was convenient, since it eliminated the need to assemble and test the circuit under sterile conditions, and the use of ethylene oxide gas as a sterilizing agent made it possible to sterilize the interior of the closed circuit at room temperature. Sterilization was always achieved, and, since the circuit remained closed after sterilization, subsequent contamination of the interior did not occur.

SUMMARY

An extracorporeal unit is described in which the pump drew upon an arterial reservoir so that its output was within broad limits independent of the venous return.

This type of pump was thought to possess advantages both for whole-body and regional perfusions since: (1) the output could be kept constant in spite of fluctuations in the venous return, due, for example, to hæmorrhage, and (2) the output could be altered easily and precisely irrespective of the venous return.

Mention is made of the sterilization of the assembled circuit with ethylene oxide gas which was found to be simple and reliable.

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THE TREATMENT OF PERFORATED DUODENAL ULCER IN THE PRESENCE OF DIAPHRAGMATIC HERNIA

By K. L. G. MILLS, P. L. BRUNNEN, and C. G. CLARK

FROM THE ABERDEEN ROYAL INFIRMARY

DIAPHRAGMATIC herniæ are common, especially those involving the hiatus, and not infrequently the latter are associated with peptic ulcer. Hæmorrhage may occur in both conditions and it may be difficult to ascertain which lesion is primarily responsible if both are present in the same patient. It is remarkable that the other major complication of peptic ulcer—namely, perforation—has rarely been reported in association with diaphragmatic hernia. Such a combination can lead to serious diagnostic difficulties and delay in treatment undoubtedly increases the gravity of the prognosis. We have been unable to find any report of diaphragmatic hernia complicated by perforated duodenal ulcer and we record our experience with 2 recent cases.

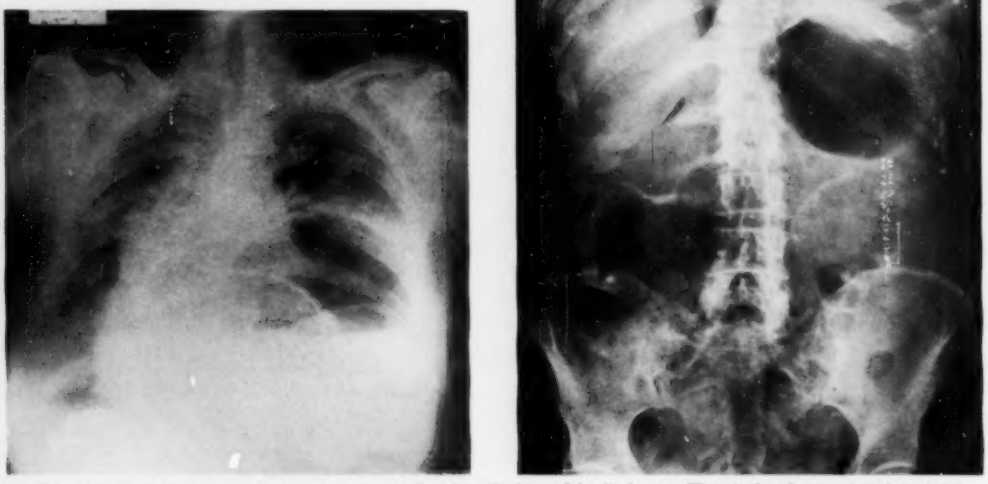


FIG. 281.—There is evidence of pneumoperitoneum below the right dome of the diaphragm. There is also a large gas bubble in the left chest, which has a sharply defined convex margin. If this is not an eventration of the diaphragm then there is a strong possibility of this being gas in a hernial sac. The presence of several grossly distended loops of bowel overlying the heart suggests the presence of a diaphragmatic hernia. The mediastinum is displaced to the right.

CASE REPORTS

Case 1.—Mrs. M. T., an obese female, first attended the out-patient department in 1951, complaining of epigastric fullness, flatulent fatty dyspepsia, and diarrhœa. Barium studies and cholecystography were carried out with negative results. The patient was lost sight of until 1956, when she attended with a new complaint of pain in the left hypochondrium for a year. A further barium meal now showed a hernia through the central tendon of the diaphragm on the left side, with displacement of the transverse colon. Conservative management was adopted and she remained well for a year, when she began to be troubled by recurrent bouts of vomiting. Barium meal now showed that part of the stomach had entered the hernial sac. The patient was unwilling to be treated surgically. With regular alkaline powders, the avoidance of bending or stooping, and sleeping with her head well propped up, she remained in good health for a further two years. It should be noted that none of the barium studies showed any evidence of peptic ulcer.

In October, 1959, the patient, now aged 70 years, was admitted as an emergency with a history of severe epigastric pain radiating over the left side of the chest. This had been present for 24 hr. On examination she was obviously shocked and dyspnœic, with evidence of generalized peritonitis. Supportive measures were instituted and radiography performed. The radiographs (*Fig.* 281) showed displacement of the mediastinum to the right, a large diaphragmatic hernia, and free gas both above and below the diaphragm. This was interpreted as perforation of a viscus, probably stomach, resulting from strangulation in the diaphragmatic hernia.

An immediate thoracolaparotomy was performed, when it was confirmed that a hernia of the central tendon of the diaphragm was present, containing part of the transverse colon, the whole of the stomach, and part of the bulb of the duodenum. The gap in the diaphragm was enlarged and the contents of the hernial sac reduced. The last portion to reduce was the duodenum. An ulcer on its anterior wall had perforated. This was repaired by simple suture. The patient's condition now gave rise to some anxiety and the operation was rapidly completed by closing the wound and draining the left pleural cavity. No attempt was made at a formal repair of the hernia;

indeed the neck of the hernia was quickly enlarged by incision and resuture in the hope of avoiding subsequent strangulation.

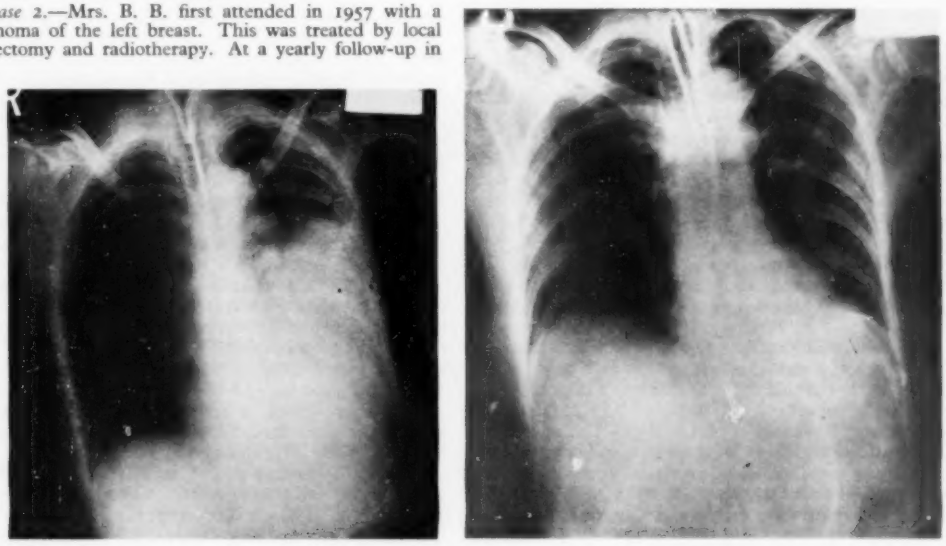
She made a good recovery from operation, being discharged from hospital six weeks later. She has been seen from time to time for review. Residual dyspeptic symptoms give her little trouble. Her chief complaint is of dyspnœa, particularly on bending. This is probably due to the very large hernia. Re-operation to reduce and repair the hernia has been suggested to the patient, but she is 71 years old, recalls her last period in hospital with some distaste, and since she is not unduly incapacitated she is averse to this suggestion.

Case 2.—Mrs. B. B. first attended in 1957 with a carcinoma of the left breast. This was treated by local mastectomy and radiotherapy. At a yearly follow-up in November, 1959, she complained of increasingly severe dyspeptic symptoms which had first appeared about five years before. There was no evidence of recurrence of her carcinoma, but routine X-ray examination of the chest showed healed fractures of the 6th, 7th, and 8th ribs on the left side. There was no evidence that these were pathological fractures, and no signs of bony metastases were observed in radiographs of the vertebræ or pelvis. On further questioning it seemed likely that the fractures had resulted from a fall in the home some five months previously. Her dyspepsia was investigated by barium meal with Trendelenburg tilt and cholecystography. In addition to an active duodenal ulcer, several gallstones were noted. She accepted the advice to submit to operation and her name was put on the waiting list.

In April, 1960, she was admitted as an emergency with a history of severe epigastric pain and vomiting for a week. On examination her general condition was satisfactory, and her temperature elevated to 100° F. There was dullness to percussion, with diminished breath-sounds at the left chest posteriorly. The upper abdomen was rigid, particularly on the right side, though the lower abdomen could be easily palpated. A diagnosis of left pleural effusion with involvement of the diaphragm was considered, or alternatively, acute cholecystitis complicated by pneumonia. Radiography of the chest (*Fig.* 282 A) was difficult to interpret, but appeared to support the first diagnosis. When the patient was re-examined 4 hours after admission it was evident that she now had a generalized peritonitis and laparotomy was indicated.

Through a right paracentral incision a perforated duodenal ulcer was seen. The duodenum was stretched over to the left side of the vertebral column due to displacement of the whole of the stomach and part of the transverse colon through a gap in the central tendon of the diaphragm. There was moderate peritoneal contamination. The ulcer was sutured, the gap in the diaphragm enlarged, and the stomach and colon were reduced. The stomach contained almost 2 litres of fluid, but there was little or no contamination of the pleural cavity (despite the fact that no peritoneal sac could be demonstrated). The pleura was drained and the lung when re-expanded appeared to be normal (*Fig.* 282 B). The gap in the tendon of the diaphragm was repaired with interrupted silk sutures and the abdomen closed.

Her immediate convalescence was satisfactory and radiography of the chest on the third post-operative day showed normal lung fields. It became apparent when oral fluids were started, however, that some degree of pyloric obstruction was present. On the seventh post-operative day a posterior gastro-enterostomy was performed to relieve this and thereafter she made a rapid recovery. She was again seen in July, 1960, in excellent health and now awaits admission for cholecystectomy which it is proposed to carry out later in the year.



A B

FIG. 282.—A, A dense opacity is present in the left hemi-thorax with a convex superior margin, the left dome of the diaphragm being obscured. There is a striking absence of gas in the left hypochrondrium. Neither the stomach gas bubble nor the splenic flexure can be identified. The appearances might be due to a large diaphragmatic hernia, although it is unusual that no bowel shadows are seen. B, Post-operative chest film.

DISCUSSION

These records show that the clinical features of perforated peptic ulcer in the presence of a diaphragmatic hernia may be difficult to analyse. Radiography in *Case* 1 pointed to the correct diagnosis but was misinterpreted in *Case* 2. The condition is rare and we have been unable to trace any report of perforated

duodenal ulcer in association with diaphragmatic hernia.

Zarowitz and Grayzel (1950) describe the autopsy findings in a patient known to have had a diaphragmatic hernia for 23 years, who died 42 hr. after the onset of sudden abdominal pain. A perforation was present in an attenuated sacculated portion of the stomach situated 4 cm. below the cardia. The stomach was distended and lay entirely within the abdomen, though it was easily dislocated into a large para-œsophageal hernial sac. Post-mortem autolysis obscured histological details, though considerable venous congestion was evident in the immediate vicinity of the gastric perforation. It remains speculative whether the perforation resulted from a peptic ulcer, or from rupture of a portion of the stomach which might have been strangulated within the hernia, only to reduce itself after death.

Kleitsch (1952) describes a patient who died 24 hr. after operation for a perforated gastric ulcer which was situated within a para-œsophageal hiatus hernia just above the point of maximum constriction of the stomach by the diaphragm. It is of interest to note that the initial diagnosis was one of left lower lobe pneumonia and laparotomy was delayed for 48 hr., when further radiographs demonstrated for the first time the presence of gas within the hernial sac.

Ochsner and Ochsner (1958) describe a female, aged 79 years, who was known to have a hiatus hernia three years previously. She had suffered from indigestion for several months and was admitted to hospital for observation following a hæmatemesis. Her condition suddenly deteriorated and at operation the hernia was found to contain the completely obstructed antral portion of the stomach, while the fundus and cardia lay below the diaphragm. There was a perforated ulcer on the greater curvature, and after the stomach was freed a wedge resection was performed and the hernia repaired. The patient survived.

These three patients all had acquired hernia, as did our *Case* 1. In our *Case* 2 a traumatic hernia secondary to the fractured ribs is assumed, and this is supported by the absence of a peritoneal sac covering the abdominal viscera. Watkins, Harper, and Condon (1952) give details of a patient who developed a strangulated traumatic hernia one year after a Smithwick sympathectomy for hypertension. The clinical picture was similar to that of our second patient and recovery followed early operation with resection of a perforated gangrenous area of the stomach. Johnston and Twente (1952) describe an interesting patient who, four days after an automobile accident in which he sustained multiple rib fractures, was found to have an acute perforated gastric ulcer. The ulcer was situated at the point of maximum constriction of the stomach by the diaphragm and the authors suggested that this allowed a fixed area of the stomach mucosa to be constantly bathed in gastric juice, with ulceration and perforation after a short interval. The patient died after operation for a thoracogastric fistula, the correct diagnosis being missed. Frank and Hamilton (1942) also describe a case of perforated gastric ulcer associated with traumatic diaphragmatic hernia. Radley and Goldsmith (1949) describe a young male who survived operation for a perforated gastric ulcer associated with a right-sided diaphragmatic hernia of congenital origin.

In all the cases described the diagnosis was not immediately apparent. In this unusual situation we might expect the mortality to be related to the delay in operative intervention, and the correct approach may be a matter of individual preference or circumstance. A perforated gastric ulcer within an obstructed hernial sac may present with few abdominal signs, and unless free gas is observed in an unusual situation the correct diagnosis may be easily delayed. It should always be considered, however, in any patient known to have a hernia and who presents with shock out of proportion to the presenting clinical signs. When the perforated ulcer is duodenal in situation, diagnosis is easier if the possibility is considered. Certainly spreading peritonitis should indicate exploratory operation. It should be noted that though radiographs are helpful, they may require expert opinion in these cases. The latter is not always readily available for an emergency service and the surgeon may have to rely on his own experience. Having reached a provisional diagnosis the correct surgical approach must be considered.

Watkins and others (1952) quote Harrington (1940) that this problem should be dealt with by a thoracic approach, since viscera prolapsing through the diaphragm lose their "rights of residence". There are, however, two features which favour the abdominal approach in the presence of a perforated peptic ulcer. Even under these exceptional circumstances the ulcer should probably be treated on its own merits. While a perforated duodenal ulcer is often dealt with by simple suture, most surgeons prefer to deal with perforated gastric ulcer by immediate gastrectomy. The decision to prolong operative time by performing partial gastrectomy, however, may be a difficult one and clearly depends on the patient's general condition. Secondly, if the hernia is acquired there will generally be a peritoneal sac to protect the pleural cavity from contamination by gastric contents, and no real purpose is offered by entering the chest. This argument does not necessarily hold in traumatic hernia, but even here if the ulcer is duodenal the pleural cavity may be protected by obstruction in the hernial orifice due to impacted intestine. In any event the pleural cavity can be readily cleared from the abdominal aspect and intercostal drainage easily effected. No harm appears to have resulted from the contamination of the pleura in *Case* 2. The degree of contamination, however, was minimal and the intestinal contents probably sterile. Both our patients were treated with penicillin post-operatively.

SUMMARY

The unusual problem of perforated peptic ulcer coexisting with diaphragmatic hernia is discussed. Two unique cases of perforated duodenal ulcer are described, one associated with acquired and the other with traumatic diaphragmatic hernia. Diagnostic difficulties are emphasized and the surgical approach is considered. Reasons are given for favouring an abdominal operation in preference to the previously suggested thoracic approach, though this is to be judged in the light of the considerable diagnostic difficulties in any individual case.

We wish to thank Mr. N. J. Logie for permission to publish details of *Case* 2. We are grateful to Dr. L. A. Gillanders for his reports on the radiograph.

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DUODENO-COLIC FISTULA DUE TO GALL-STONES

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A RECENT survey of benign duodeno-colic fistulæ resulting from a variety of causes has been made by Grayson and O'Connell (1960). The total number of such cases which they are able to find recorded in any detail is small, whilst benign duodeno-colic fistulæ due to biliary tract disease prove to be rarities.

Murchison (1857) is the first to mention a case where a fistulous communication existed between duodenum and colon through the gall-bladder, while Judd and Burden (1925) mention 4 similar cases, but no details of any of these cases are given. In the case of duodeno-colic fistula reported by Neville (1954), the gall-bladder was found to contain stones but the evidence that the cause of the fistula was biliary is only presumptive as no fistula was found in the gall-bladder.

In fact, the only 2 previously well-documented cases of duodeno-colic fistula due to cholecystitis are those of Lovell (1947) and of Rosenquist and Sjoberg (1955).

A further case of duodeno-colic fistula due to gall-stones is presented here.

CASE REPORT

First Admission.—A woman of 68 years was admitted in August, 1955, complaining of severe pain in the right hypochondrium, going through to the back, of three days' duration together with vomiting. There was a previous history of indigestion especially after fatty foods for many years. There was no history of jaundice.

ON EXAMINATION.—The patient had a pyrexia of 103° F. but there was no clinical jaundice. Apart from marked tenderness in the right hypochondrium, there were no abnormal physical signs.

RADIOLOGICAL FINDINGS.—At that time a plain radiograph of the abdomen showed the presence of two large laminated stones in the right upper quadrant (*Fig.* 283). A cholecystogram six weeks later still showed these calculi but failed to show any evidence of gall-bladder concentration.

A barium meal and follow-through failed to reveal any lesion of the upper gastro-intestinal tract and progress of the barium at twenty-four hours was normal. The duodenal cap was normal and showed no evidence of peptic ulcer.

PROGRESS.—The patient's pain subsided satisfactorily on a conservative régime.

Because of her age and frail physical appearance, no operation was advised and she was discharged home.

SECOND ADMISSION.—The patient was re-admitted in November, 1959, now aged 72 years. She had continued to have indigestion after fatty foods ever since her previous admission four years previously but had suffered no severe pain until three days earlier. The severe pain was again situated in the right hypochondrium and was accompanied by vomiting. The vomiting was not severe, however, and was at no time fæcal. Her bowels had been open regularly every day with no history of diarrhœa. There was still no history of jaundice.

ON EXAMINATION.—The patient was apyrexial and there was no evidence of jaundice, weight-loss, or severe dehydration. Apart from some epigastric tenderness, there were no abnormal physical signs in the abdomen.

RADIOLOGICAL FINDINGS.—At this time her radiological investigation consisted of a plain radiograph of abdomen, barium enema, and barium meal, in this order. The plain radiograph failed to show the presence of the previous gall-stones. There was no evidence of any obstructive lesion or gall-stone ileus, but air was present in the biliary tract.

In view of the lower abdominal pain and tenderness in the left iliac fossa at this time, it was felt that a barium enema should be the first investigation. This revealed a normal colon apart from the track of barium from near the hepatic flexure towards the air in the biliary tract (*Fig.* 284).

The large sac of air communicating with the duct air was at that time thought to be in the gall-bladder, but there was no reflux into the duodenum or adjacent small bowel.

It was felt that a barium meal would now assist. This showed a normal stomach with no evidence of pyloric delay. It was noted that a track of barium passed from just near the apex of the duodenal cap towards the air in the biliary tract. The large sac of air previously seen on the enema was not now present and one presumes that this had been in the pyloric end of the stomach and duodenal cap. A second fistula, much better filled than on the enema film, was also seen passing down and to the right into the region of the previous fistula from the colon (*Fig.* 285).

It was felt, in view of the disappearance of the previously demonstrated gall-stones and the later radiological findings, that a fistula was present between the gall-bladder and duodenum and between the gall-bladder and colon.

OPERATIVE FINDINGS.—At laparotomy, dissection in the region of the gall-bladder was made difficult by adhesions. The identification of the gall-bladder was not easy at first and was greatly assisted by an operative cholangiogram performed by needle aspiration of the common hepatic duct. This cholangiogram showed the flow of dye into the duodenum to be normal. The

gall-bladder was then found to be no more than a narrow fibrous tunnel lying vertically across both the first part of the duodenum and the transverse colon immediately distal to the hepatic flexure, the duodenum and colon having been drawn closely together by fibrous contracture. The gall-bladder contained no stones but communicated with both duodenum and colon by two separate fistulæ, the duodenal fistula being the larger of the two.

The fibrosed gall-bladder was dissected free from duodenum and colon and removed. The duodenum and colon were then separated, their fistulæ excised and closed.

POST-OPERATIVE COURSE.—The patient had an uncomplicated post-operative recovery, and, when seen at routine follow-up twelve months later, was feeling well and was free from symptoms.

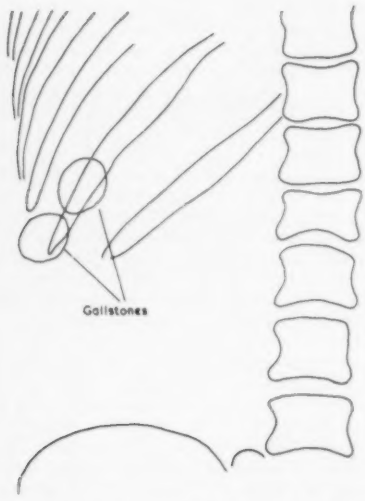

FIG. 283.—August, 1955: Two large laminated gall-stones are seen on straight radiographic examination of the abdomen.

FIG. 284.—November, 1959: A barium enema reveals a fistula arising in the hepatic flexure of the colon. Gas is present in the bile-ducts, but there is no evidence of any opaque gall-stones.

DISCUSSION

For what length of time this patient's duodeno-colic fistula had existed it is impossible to say. The only severe attack of pain experienced by her prior to her re-admission to hospital was four years previously, and it had been shown that the gall-stones were still in situ six weeks after this attack had subsided. It must be presumed that the stones ulcerated through into the duodenum and colon quite insidiously without giving rise to any major symptoms.

The symptoms produced by the duodeno-colic fistula in this case were not as severe as might have been expected. Fæcal vomiting was a prominent feature of the case described by Lovell (1947), while watery diarrhœa and emaciation, the result of jejunal irritation by colonic content, were the principal symptoms in the case reported by Rosenquist and Sjoberg (1955). Neither of these major symptoms was found in the case presented here, the diagnosis of the fistula being essentially a radiological one.

SUMMARY

A case of benign duodeno-colic fistula through the gall-bladder is presented, thought to be due to the ulceration of gall-stones into both duodenum and colon. The absence of major symptoms is noted, the diagnosis being a radiological one.

FIG. 285.—November, 1959: A barium meal shows a fistula between the duodenum and the colon. Gas is again present in the bile-ducts.

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TEN-YEAR RESULTS OF VAGOTOMY AND GASTROJEJUNOSTOMY IN THE TREATMENT OF CHRONIC DUODENAL ULCER

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SURGICAL opinion is still divided as to the best operation for the treatment of chronic duodenal ulcer. Sub-total gastrectomy has for many years remained the most popular operation, and not without good reason, because it has produced results far superior to those obtainable from the more conservative operations in vogue when it was introduced. Experience has also shown that a minority of patients after a sub-total gastrectomy suffer from side-effects either in the form of nutritional deficiencies or the various post-cibal syndromes. It is against this background of a well-established and, on the whole, a very successful operation that any alternative procedure must be considered. The most promising of these operations is a division of the vagus nerves combined with some procedure which enables the stomach to

empty freely. The drainage operation employed may be a posterior gastrojejunostomy, a resection of the pyloric antrum, a pyloroplasty, or a resection of the anterior half of the pyloric sphincter. Which of these combined procedures will give the best results is still uncertain since some of them have to pass the test of time. In an earlier paper (Holt and Robinson, 1955) the short-term results of vagotomy and gastrojejunostomy were reported and it was found that this operation provides protection from recurrent ulceration at least equal to that following gastrectomy, and appears to be followed by a lower incidence of troublesome side-effects. This experience has not been universal and some surgeons report a high incidence of recurrent ulceration following vagotomy and gastrojejunostomy. The reluctance of many surgeons to accept vagotomy and some drainage procedure as a sensible treatment of duodenal ulcer is based largely on a suspicion that the long-term results may not be so good, and that some unspecified sequelæ are bound to appear.

So far there have been few published reports of the long-term results of vagotomy in the treatment of chronic duodenal ulcer and none to our knowledge in which the follow-up has been complete. The purpose of this article is to provide some evidence which may assist surgeons to assess the value of vagotomy and gastrojejunostomy in the treatment of duodenal ulcer. This combined operation was selected originally because it appeared to be the safest and simplest method of overcoming the gastric retention which follows vagotomy. The results we have obtained give ample proof of the contention that as regards mortality and freedom from recurrent ulceration this operation is better than any other, but there is no denying that some of the sequelæ which are troublesome after gastrectomy are found also, though perhaps to a less degree, after vagotomy and gastrojejunostomy.

MATERIAL

In a previous publication the results of a series of 243 patients with chronic duodenal ulceration treated by vagotomy and gastrojejunostomy were described in detail (Holt and Robinson, 1955). The first 100 of these cases whose operations were performed before July, 1950, form the basis of this report. The cases are consecutive in that all cases of duodenal ulcer, excluding acute perforation and severe hæmorrhage, accepted at that time by one of us (R. L. H.) for surgical treatment, were treated by this procedure. There was no attempt to segregate the patients according to age, sex, the degree of gastric hypersecretion, or any other factor, into groups which might receive different treatment. The follow-up has been complete in that every patient has been traced and the results have been assessed severely, using the grading of Visick (1948) which is interpreted as follows:—

Grade I: Patients who are symptom-free, enjoying a full diet, not requiring medicines, working full time, and completely satisfied with the results of the operation.

Grade II: Patients who have mild gastro-intestinal complaints which are easily controlled, who are working full time, and are satisfied with the results of the operation.

Grade IIIS: Patients who have mild residual symptoms which are not fully controlled by simple precautions. These patients are working and do not find their symptoms a hindrance.

Grade IIIU: Patients with residual symptoms who are not satisfied with the result of their operation or patients considered by us to have an unsatisfactory result.

Grade IV: Not improved.

A simple tabulation of the over-all results after 10–13 years will not necessarily give a complete picture. A patient who has been symptom-free for 9 years and then develops a recurrent ulcer may appear in Grade IV, whereas one who has severe persistent symptoms for 9 years and then goes into remission may appear in Grade I. In order to overcome this difficulty Pulvertaft (1958) in assessing the results of gastrectomy uses a 'mean' grade which represents the average grades recorded at each annual follow-up since the operation. We have not used this method, because in our experience it is rare for a patient who does badly in the early years after operation to undergo spontaneous improvement. If after an initially favourable result a patient relapses, then in our opinion the operation should be regarded as a failure. We have therefore recorded the Grade at the time of the most recent assessment, i.e., 10–13 years after operation. The only exception to this has been in the case of 3 patients who required further gastric operations, and these have been assessed at their Grade immediately before the second operation.

RESULTS

These are summarized in the accompanying tables. In the 100 patients reviewed there were no operative deaths and only 1 case of recurrent ulceration. (In over 600 cases the mortality-rate is 0·5 per cent and the low incidence of recurrent ulceration continues.) Sixteen of the 100 patients under review had died since operation from causes listed in *Table I*. These deaths were unrelated to the original operation with the possible exception of *Case* 9. This patient died seven years after his operation, shortly after admission to a neighbouring hospital and before investigations were complete. There had been a history of recent vomiting for

Table I.—Deaths since Operation—16 Patients

Males	Interval from Operation to Death	Cause of Death
1	5 mth.	Coronary thrombosis
2	7 mth.	Pemphigus and bronchopneumonia
3	9 mth.	Glioblastoma multiforme (cerebral)
4	2 yr.	Acute gastro-enteritis
5	2 yr.	Carcinoma, rectum
6	3 yr.	Acute enteritis
7	4 yr.	Miliary tuberculosis
8	5 yr.	Myelomatosis
9	7 yr.	Uræmia due to pyloric stenosis
10	8 yr.	Carcinoma, lung
11	8 yr.	Carcinoma, bronchus
12	9 yr.	Cerebral hæmorrhage
13	9 yr.	Myocardial infarction
14	9 yr.	Cerebral hæmorrhage
15	9 yr.	Coronary thrombosis
16	10 yr.	Carcinoma, œsophagus

several months before admission to hospital and the certified cause of death was uræmia due to pyloric stenosis. Autopsy was not performed.

Table II.—Over-all 10-year Results—84 Cases

Visick's Grading	Males	Females	Total	Remarks
I No symptoms	31	4	35	
II Mild symptoms easily controlled	32	4	36	90·5 per cent satisfactory
IIIS Mild symptoms not controlled but satisfactory	2	3	5	
IIIU As IIIS, but unsatisfactory	2	1	3	9·5 per cent unsatisfactory
IV Not improved	4	1	5	

The over-all results in the 84 survivors are given in *Table II*. Included in these 84 are 3 patients who required further surgery and are assessed as unsatisfactory. Of these one (Grade IV) underwent a partial gastrectomy for hæmatemesis due to stomal ulcer 4 years after vagotomy. The other two (Grade IIIU) were operated on for bilious vomiting 6 and 11 years respectively after the vagotomy and gastrojejunostomy. In both cases the gastrojejunostomy was separated and an anterior pylorectomy performed with such improvement that both patients are now regarded as satisfactory results. It is quite evident from this Table that the operation has withstood the test of time in a most encouraging manner; 90 per cent of the patients are assessed as having obtained a satisfactory result. This view is supported by *Tables III* and *IV* which show the high percentage of patients fully employed and able to maintain their body-weight and energy.

Table III.—Ability to Work

Working full time—Original job	53
Working full time—New job	12
Retired	8
Housewives	6
Working part time (female)	1
Not working	3
No information	1

UNSATISFACTORY RESULTS

These fall into two groups. The first group can be blamed on the shortcomings of the operation itself, and the second on bad selection of cases. The symptoms of the unsatisfactory cases are listed in *Table V*. Apart from 1 case of stomal ulceration no other unsatisfactory result can be blamed on a single factor but rather on a combination of factors, none of them singly severe, which when present together have made the patient miserable. The most troublesome complaint has been bilious vomiting which resulted in 3 patients having further operative treatment as previously mentioned. *Case* 4 who has complained of persistent ulcer pain has nevertheless continued full-time working at his old occupation, does not consider his pain bad enough to warrant further surgery, and several radiographic examinations have failed to reveal any evidence of recurrent ulceration. *Case* 5 was discovered afterwards to have undergone psychiatric treatment and had received several courses of E.C.T. Each time his mental condition relapses he suffers a recurrence of his gastric symptoms. *Case* 8 is definitely menopausal and in *Case* 6 the persistent symptoms were due to a subcostal neuritis which was ultimately treated by division of the subcostal nerve.

Table IV.—Weight

Gained since operation	28
Unchanged	39
Lost since operation	15
No information	2
Total	84

Table V.—Analysis of Unsatisfactory Results

Males	Age	Symptoms	Ability to Work	Time of Relapse
IIIU 1	43	Bilious vomiting. Converted to pylorectomy at 6 yr. Five years later has occasional vomiting and flatulence only	Lost time	4 yr.
2	30	Sub-sternal pain p.c. Vomiting. Barium meal—possible intussusception at stoma	Light work	2 yr.
IV 3	34	Hæmatemesis, stomal ulcer—partial gastrectomy performed		4 yr.
4	48	Ulcer pain persists. Occasional vomiting. Barium meal at 10 yr. shows scarred duodenum but no stomal ulcer	Full time	From beginning
5	30	Pain, vomiting, and dumping during episodes of depression. Repeated courses of E.C.T. Barium meal—no recurrence at 9 yr.	Lost time	2 yr.
6	33	Sub-costal pain not due to ulcer. Vomiting and dumping	Not working	From beginning
Females				
IIIU 7	51	Bilious vomiting and diarrhœa. Converted to pylorectomy at 11 yr. Improved	Housework	From beginning
IV 8	40	Pain, nausea, dumping, fullness. Menopausal. Barium meal—no recurrence at 8 yr.	Housework	From beginning

RESIDUAL SYMPTOMS

A totally erroneous impression would be obtained of the results of this operation if investigation were limited to a consideration of the over-all and unsatisfactory results. There is no doubt about the value of the operation in curing the original ulcer and in preventing recurrent ulceration. The frankly unsuccessful results which can be fairly blamed on the operation itself are few, but if we inquire carefully into the incidence of mild degrees of post-operative abdominal disturbances we find them present in a considerable proportion of the patients. A word of warning is necessary here. A careful follow-up with persistent questioning ends by eliciting symptoms which are transient, mild, rare in their occurrence, and in fact are no more than one would expect to find in a cross-section of normal healthy individuals. Occurring in a patient who has had a gastric operation they tend to be blamed on the operation. A detailed record of these complaints is found in *Table VI*. At first it might seem difficult to accept this high incidence of residual symptoms persisting after an operation which is claimed to be 90 per cent successful. It is only when the patients' complaints are carefully considered that the true picture is revealed.

Table VI.—Residual Symptoms—83 Cases*

Symptoms	Number	Per Cent
'Fullness' p.c.	40	48
Slight	17	20
More troublesome	23	28
Vomiting	19	23
Occasional or slight	15	18
More frequent or severe	4†	5
Diarrhœa	16	20
Mild	8	10
More severe	8	10
Pain	9	11
Dumping	5	6

* Patient subjected to gastrectomy excluded.
† Two improved by further operation.

A sensation of uncomfortable fullness after meals is the commonest complaint. This, on inquiry, is frequently found to be self-inflicted and is simply due to over-eating. To some extent this is understandable in a patient who not infrequently has been kept on a dull ulcer diet for years. A second factor is that we believe that patients after vagotomy have a smaller appetite than before operation. In other patients the fullness seems to be brought on by a particular article of diet, e.g., fats, sweets, milk.

The second commonest complaint is vomiting of bile particularly on waking in the morning and usually before taking any food. In the majority of our cases the vomiting was trivial and occasional, but in 3 cases it was adjudged to be more troublesome and was treated by undoing the gastrojejunostomy and excising the anterior half of the pyloric sphincter with good results. It has been noticeable, and rather difficult to explain, that many of our patients who complain of some abdominal discomforts when at home promptly lose them as soon as they are admitted to hospital. This applies particularly to vomiting, fullness after meals, and diarrhœa. A full ordinary diet in hospital is taken with apparent enjoyment and no ill-effects.

The next complaint in order of frequency is diarrhœa. A lot has been written about this particular complication and in our opinion it has been very much over-stressed. It is true that when directly questioned 20 per cent of our patients admit to having had some diarrhœa but in no case was any patient severely troubled by it. Almost as many patients state that they are more constipated after operation than before. It is unusual for diarrhœa to begin whilst the patient is in hospital. In quite a considerable proportion of patients the first attack begins acutely some time after the patient has returned home, is present for only short spells, and there are long free periods. Many of the patients are constipated between the attacks of diarrhœa. Only a few of these cases of diarrhœa can be blamed on the operation itself; many are suffering from a recurrent enteritis or enterocolitis which has become quite a common condition in this country since the war.

The incidence of pain similar to the patient's ulcer pain has not been very marked. It usually responds readily to alkalis and all patients with this complaint have been investigated and in no case has any evidence of further ulceration been revealed or suggested.

Finally the dumping syndrome has not proved to be a frequent or troublesome complication. Perhaps this has been due to the fact that we have aimed at making the gastrojejunostomy stoma rather small.

DISCUSSION

When Dragstedt (1945) reintroduced the operation of vagotomy as a method of treating duodenal ulcer surgeons were treating this condition by a partial gastrectomy or posterior gastrojejunostomy. At that time the success or failure of the operative treatment of duodenal ulcer was judged by the incidence of subsequent stomal ulceration or recurrent duodenal ulceration. In this respect gastrectomy gave very satisfactory results, providing at least three-quarters of the stomach was excised. This extensive resection and the subsequent anastomosis of the stomach to the jejunum led in some cases to troublesome post-cibal symptoms. The problem has been to find another surgical procedure which gave the patient as good protection from recurrent ulceration as did gastrectomy and which left the patient less liable to troublesome sequelæ. As is well known the early experiments in which a vagotomy alone was carried out in the treatment of duodenal ulcer were very unsatisfactory because a large percentage of the patients developed gastric retention. The interest of those surgeons who thought vagotomy a sensible approach to the treatment of duodenal ulcer has subsequently been concentrated on finding the best method of overcoming this gastric retention. The operation most frequently combined with vagotomy has been a posterior gastrojejunostomy. There was much to recommend this combination at that time. It was an operation with a very low mortality, and it was thought that, by itself, it

frequently cured duodenal ulcer. The results of this combined operation have been published by numerous authors. Most of these papers have reported on the short-term results and have been concerned chiefly with the incidence of recurrent ulceration. This has varied widely. Pallette and Harrington (1958) in a 10-year follow-up of 58 cases, found an 8·6 per cent incidence of stomal ulceration. Walters and Mobley (1957) found a 13 per cent recurrent ulceration at the Mayo Clinic.

Table VII.—Comparison of the Authors' Results with Those Reported by Stammers (1959)

Results	Gastrectomy	Vagotomy and Gastro-jejunostomy
Operative mortality	2–3 per cent	Nil
Cured or greatly improved	70 per cent	84 per cent
Considerably better	15 per cent	6 per cent
Unsatisfactory	10–12 per cent	10 per cent

In this country Henson and Rob (1955) reported a recurrence rate of 12·4 per cent in a group of cases followed only for 29 months. Oberhelman and Dragstedt (1955) record stomal ulceration in 5·8 per cent of 487 cases followed for between 2 and 12 years. Burge (1960) in a 10-year follow-up of 301 cases found a 3·8 per cent incidence of anastomotic ulcer amongst 262 survivors whilst Feggetter (1959), in 600 cases followed up from 1 to 5 years, found stomal ulceration in only 1 per cent and recurrent duodenal ulceration in less than 1 per cent. Obviously there are wide divergences in the results published, and they are difficult to explain. We have never been in doubt about the efficacy of vagotomy and gastrojejunostomy in curing duodenal ulceration and preventing stomal ulceration. We have used this operation in over 600 cases with only 4 known cases of subsequent ulceration or hæmorrhage. In the group reported in this paper all the patients have been traced, they represent the first 100 of our patients treated by this method for duodenal ulcer, and all have been followed-up for 10 years or more. We do not expect any higher incidence of stomal ulceration or hæmorrhage in the remaining 500 patients, practically all of whom were operated on more than 5 years ago. In our hands this operation has been very safe and proved most satisfactory in curing duodenal ulcer and preventing secondary ulceration. Our results compare favourably with those obtained from partial gastrectomy. In *Table VII* our own results are compared with those reported by Stammers (1959) as representing the average results of gastrectomy from several large centres.

The operation has not been so successful in preventing the occurrence of post-cibal symptoms, though Cox and Kerr (1957) reported that these were slightly less after vagotomy and gastrojejunostomy than after partial gastrectomy. They found the incidence of bilious vomiting to be the same after each operation. Dumping was slightly less common and the nutritional disturbances were significantly less common in the vagotomy group. They found that more patients were able to take a normal-sized meal, more could take a sweet course, and fewer had selective impairment of digestion after vagotomy and gastrojejunostomy. There has been a widespread tendency to blame these disturbances on the vagotomy, but in our opinion there is an extraordinary lack of evidence to support this view. These symptoms occur after any operation in which the stomach is anastomosed to the jejunum and the duodenum by-passed.

A wealth of evidence is accumulating in support of the suggestion that many of these symptoms are due to the rapid emptying of the stomach into the jejunum. This is obviously more likely when the gastric stump is small and the anastomosis to the jejunum wide. A second factor which is important in patients who have trouble in maintaining their energy and weight is the imperfect digestion which follows on the loss of the gastric phase of digestion and the passage of food into the jejunum ahead of the secretions from the pancreas and liver. There is no need to go into great detail since all the evidence points towards avoiding whenever possible an anastomosis between the stomach and jejunum.

We have re-operated on 3 of our patients who had troublesome post-cibal symptoms or a combination of these with bilious vomiting. We undid the gastrojejunostomy and restored the continuity of the gastro-intestinal tract. To get over the gastric retention which would otherwise have followed this procedure we excised the anterior half of the pyloric sphincter somewhat after the manner described by Judd and Nagel in 1927 and later used by Beattie (1950).

In every case there was a marked diminution in the patient's symptoms and all of them can now be upgraded into the satisfactory results. Since then we have adopted this combination of vagotomy and anterior pylorectomy as the operation of choice in the treatment of duodenal ulceration, but in the rare case in which technical difficulties might make an anterior pylorectomy difficult or even dangerous we have no hesitation in carrying out the old combination of vagotomy and gastrojejunostomy.

Bruce, Card, Marks, and Sircus (1959) have adopted a more selective approach to the treatment of duodenal ulceration which they suggest might reasonably be based on the secretory response of the individual patient to maximal histamine stimulation. They suggest that patients in whom the acid output is not particularly high can be treated by standard gastrectomy or vagotomy combined with one of the more conservative drainage procedures, whereas more extensive operations are necessary to cure duodenal ulceration and prevent recurrent ulceration in those patients with a very high pre-operative acid output. Our experience suggests that vagotomy provides by itself adequate protection against recurrent ulceration, and that a limited gastrectomy in addition is unnecessary and adds a little further operative risk. One of the objects of the operation is to leave the patient with a digestion as near normal as possible and we feel that this is most likely to be achieved by a vagotomy combined with a resection of the anterior half of the pyloric sphincter. This operation leaves the patient with a reasonable gastric secretory response to food, a stomach which does not usually empty over-quickly, and a normal direction of food and secretions along the alimentary tract.

CONCLUSIONS

The results of 100 patients with duodenal ulcer treated by vagotomy and posterior gastrojejunostomy followed up for over 10 years are reported. The operation has proved to be extremely successful in preventing recurrent ulceration, and it has a very low operative mortality. It does not prevent the occurrence of post-cibal symptoms similar to those seen after gastrectomy though there is some evidence to suggest the incidence is lower than after gastrectomy. The authors believe that most of these post-cibal symptoms are the result of anastomosing the stomach to the jejunum, particularly with a wide stoma, and suggest that the best drainage operation to combine with vagotomy is a simple excision of the anterior half of the pyloric sphincter.

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RETROGRADE JEJUNOGASTRIC INTUSSUSCEPTION

A REVIEW OF 10 CASES DIAGNOSED RADIOLOGICALLY

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RETROGRADE intussusception of the jejunum into the stomach, through an operative stoma, is one of the less common complications of gastro-enterostomy and of Polya-type gastrectomy.

The term 'retrograde intussusception' was first introduced by Hunter in a paper read to the Society for the Improvement of Medical Knowledge (1793): "When the introsusception is downward it may be called progressive, and when it happens upwards, retrograde."

Further, discussing the causes of intussusception he said, "Introsusception is a disease produced by the passing of one portion of the intestine into another, and it is commonly, I believe, from the upper passing into the lower part. If the mode of accounting for the introsusception which I am going to offer be just, it will more frequently happen in the way I have stated, although there is no reason why it may not take place in the contrary direction, in which case the chance of cure will be increased by the natural actions of the canal tending to replace the intestine, and probably from this circumstance it may oftener occur than commonly appears."

Hunter, at this time, had not seen a case, but at the same meeting Home (1793) reported a case involving the large bowel.

Akehurst (1955) collected 103 cases, from the literature, of retrograde intussusception, in the non-operated bowel, involving all sites from the gastro-duodenal junction to the distal sigmoid.

Retrograde jejunogastric intussusception, following surgery, was first described by Bozzi (1914) and subsequently by von Steber (1917). Aleman (1948) found descriptions of 70 cases, of which 12 had been recognized radiologically. At the present time, about 130 cases have been reported, of which 40 have had radiological confirmation. It seems appropriate, therefore, to consider 10 cases which have been seen in this hospital during the preceding 10 years.

CASE REPORTS

Case 1.—This 68-year-old female had a cholecystectomy in 1949 for flatulent dyspepsia, when a gall-bladder containing multiple calculi was removed. Some dyspeptic symptoms persisted, however, and in 1951 a barium meal demonstrated an ulcer of the second part of the duodenum.

Two months later, laparotomy revealed dense adhesions around the previous operation site and it was thought unwise to carry out an extensive exploration. The stomach was therefore divided, the ends closed, and posterior gastro-enterostomy performed.

On the fifth post-operative day she started to have abdominal pain, distension, and vomiting of bile-stained fluid, which persisted for the next twelve days. During this period she was treated by gastric suction, and intravenous fluids. A barium meal at this time showed intussusception of the efferent loop through the stoma, with poor filling of the efferent loop, and gross retention of barium in the stomach at three hours; the afferent loop did not fill.

On the day following the barium meal she was greatly improved, and was discharged five days later. She is not known to have had any recurrent symptoms since that time.

Comment.—Acute retrograde intussusception, commencing five days post-operatively, with spontaneous resolution on the seventeenth post-operative day.

Case 2.—This man, aged 45 years, had a three-year history of ulcer-type dyspepsia. Several barium meals demonstrated a persistently deformed duodenal cap, with a small posterior ulcer crater.

At operation, this appearance was confirmed, and posterior gastro-enterostomy and Burge-type modified vagotomy carried out.

On the third post-operative day, he developed colicky upper abdominal pain and vomiting. Plain films of the abdomen showed distension of the stomach and afferent loop, suggestive of obstruction at the stoma.

The following day, barium meal demonstrated the characteristic intragastric tumour, with dilated afferent loop, and no filling of the efferent loop, even in a delayed

FIG. 286.—*Case* 2. Typical crescentic-shaped intragastric filling defect, showing the characteristic mucosal folds of a jejunal intussusception. Dilated afferent loop and no filling of the efferent loop.

film (*Fig.* 286). His abdomen was re-opened on the same day, but the stoma was found to be normal and no obstruction was demonstrated. Following this, he made an uninterrupted recovery.

At follow-up a year later, he still had occasional cramping abdominal pain, but no intussusception could be demonstrated.

Comment.—Acute post-operative jejunogastric intussusception. Negative findings at laparotomy, but uninterrupted recovery following it.

Case 3.—This male clerk, aged 44 years, had a two-year history of typical ulcer dyspepsia. A barium meal demonstrated a chronic duodenal ulcer. Medical treatment was instituted, but he did not respond, and a further barium meal five months later showed a persistent ulcer crater. At operation, a small anterior duodenal ulcer was found, and vagotomy and posterior gastro-enterostomy was performed.

He remained well, until the tenth post-operative day, when he had an attack of vomiting; he was thought to be better next day, and, it being Christmas Eve, he was sent home. However, vomiting of bile-stained fluid recurred, and he was re-admitted on the fifteenth post-operative day. Three days later, a barium meal demonstrated a jejunogastric intussusception, with, at first, no gastric emptying (*Fig.* 287). However, in a sixty-minute film, considerable filling of the upper small bowel had occurred, and the intussusception was no longer apparent. From this time his symptoms subsided rapidly and he was discharged three days later. A barium meal three months later showed a normally functioning stoma.

Comment.—Acute post-operative jejunal herniation, apparently undergoing reduction following barium meal.

Case 4.—Male aged 66 years. This patient had a long history of ulcer-type dyspepsia. A barium meal showed pyloric stenosis thought to be associated with duodenal ulcer, although the duodenal cap was poorly demonstrated.

At operation he was found to have severe pyloric stenosis, with dense adhesions around the first part of

FIG. 287.—*Case* 3. Intragastric filling defect, with the lumen of the jejunum seen *en face*.

the duodenum. This was considered to render exploration contra-indicated, and posterior gastro-enterostomy was performed.

On the tenth post-operative day he began to have intermittent abdominal pain accompanied by copious vomiting of bile-stained fluid. He was treated by gastric suction and intravenous fluids.

A barium meal on the fourth post-operative day showed the characteristic crescentic-margined filling defect within the stomach, with gross delay in gastric emptying, and no filling of the afferent loop.

His condition gradually improved, and barium meal on the thirtieth post-operative day showed a normally functioning anastomosis, with no delay in gastric emptying. Follow-up has not been possible, and his subsequent history is unknown.

Comment.—Acute post-operative jejunogastric intussusception with spontaneous reduction.

Case 5.—This man, then aged 32 years, first had a hæmatemesis 21 years ago. This resolved on medical treatment, but when it recurred 2 years later, gastro-enterostomy was performed. For the next 11 years he continued fairly well, but 8 years ago, he developed a further severe hæmatemesis which brought him into hospital. The acute episode subsided on medical treatment, but a barium meal demonstrated an anastomotic ulcer.

At operation a hard indurated mass was found around the gastro-enterostomy, with a large ulcer crater at the anastomosis.

The gastro-enterostomy was undone, and a Polya partial gastrectomy was carried out.

On the fifth post-operative day he developed abdominal distension, and began to vomit bile-stained fluid. This persisted until the eleventh post-operative day and was treated by gastric suction and intravenous fluid. On this day barium-meal examination showed a loop of jejunum herniated into the stomach. Neither the afferent nor efferent loop filled, and there was no evidence of gastric emptying at 30 min. (*Fig.* 288).

A year later he was symptom-free, and a barium meal at that time showed a normal Polya gastrectomy.

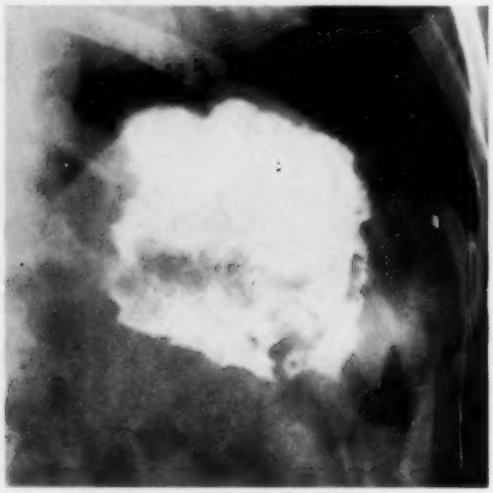

FIG. 288.—*Case* 5. Thirty-minute film showing curved filling defect. No filling of either the afferent or efferent loops.

Comment.—Acute post-operative jejunal herniation which reduced spontaneously and has not recurred.

Case 6.—This 59-year-old woman presented in 1949 with epigastric pain and vomiting of twelve months' duration, aggravated by food and unrelieved by alkalis. A cholecystogram revealed multiple calculi in a poorly functioning gall-bladder. At laparotomy, a 'strawberry' gall-bladder, with multiple surrounding adhesions, was removed.

A year later her symptoms were still persisting, and a barium meal at this time demonstrated a duodenal ulcer. At operation, dense adhesions to the gall-bladder bed were present, and a posterior gastro-enterostomy was performed.

On the fifth post-operative day she started to have colicky abdominal pain and bile-stained vomit. She was treated by gastric suction and intravenous fluids, and her symptoms gradually subsided. A barium meal on the thirteenth post-operative day revealed a typical intragastric tumour, proximal to the stoma, with dilatation of the afferent loop and retention of two-thirds of the barium at six hours (*Fig.* 289). She was discharged, improved, on the twenty-second post-operative day. A year later she was still having occasional attacks of cramping abdominal pain and vomiting, and a barium meal at this time again showed a small retrograde intussusception, though without marked delay in gastric emptying.

Comment.—Acute post-operative retrograde intussusception of the efferent loop, followed by the chronic recurrent form.

Case 7.—This 57-year-old man had a long history of dyspepsia, relieved by alkalis. A barium meal showed a moderate-sized posterior wall duodenal ulcer. Since this did not respond to medical treatment surgery was undertaken, and posterior gastro-enterostomy and Burge-type vagotomy were carried out.

On the sixth post-operative day, he developed symptoms of high intestinal obstruction, with vomiting of bile-stained fluid. He was treated by gastric suction and intravenous fluids, but the symptoms persisted.

FIG. 289.—*Case* 6. Six-hour film, showing intragastric filling defect, with considerable retention of barium, dilatation of the afferent loop, and some filling of the efferent loop.

On the tenth post-operative day, barium meal showed a circular intragastric tumour, with filling of a dilated afferent loop, but negligible filling of the efferent loop (*Fig.* 290).

Very little barium had left the stomach at 6 and 24 hours, but a film at 30 hours showed that nearly all the contrast had passed into the large bowel. From this time onwards his symptoms rapidly abated, and he was discharged on the seventeenth post-operative day.

He was seen again five months later, with a history of attacks of cramping upper abdominal pain, accompanied by bile-stained vomiting. The attacks lasted for two to three days and then passed off, to recur three or four weeks later.

A barium meal at this time showed the presence of a retrograde intussusception, which differed only from the post-operative appearances in that there was no more than slight delay in filling of the efferent loop, and gastric emptying was not markedly retarded (*Fig.* 291). He was treated with large doses of sodium bicarbonate in an effort to prevent the intussusception, but a barium meal three months later again demonstrated the jejunal herniation. On this occasion, however, it was apparent only in the supine and prone positions, and appeared to reduce spontaneously in the erect posture. His symptoms have persisted, but they are considerably less severe.

Comment.—A case of acute post-operative intussusception, which has been followed by the chronic recurrent type.

Case 8.—This 58-year-old woman had had a gastro-enterostomy for duodenal ulcer sixteen years previously.

She continued well for a number of years following this, but then began to get attacks of cramping epigastric pain lasting about half an hour. These attacks always occurred after eating starchy food; they were instantly relieved by vomiting. They tended to occur every day for a week or so, and she would then be symptom-free for two or three months.

She was admitted for investigation of these symptoms. Barium meal demonstrated an intussusception of the jejunum through the stoma, but there was only slight delay in gastric emptying. A further barium meal a few days later confirmed those findings, and it was found that the intussusception could not be reduced by palpation at the time of screening.

FIG. 290.—*Case* 7. Acute retrograde intussusception. Large intragastric filling defect. The afferent loop is dilated and there is very little filling of the efferent loop.

At operation, dense adhesions were present around the stoma, and the efferent loop was found to be intussuscepted for a length of about 4 in. The gastro-enterostomy was undone, and Billroth-I-type partial gastrectomy was carried out to restore the normal circuit. She has had no recurrence of symptoms since that time.

Comment.—Chronic recurrent retrograde intussusception sixteen years after gastro-enterostomy, the region of the anastomosis being found to be surrounded by dense adhesions.

Case 9.—This 65-year-old man had a gastro-enterostomy eighteen years previously for ulcer-type dyspepsia.

For a number of years following this he had attacks of upper abdominal pain and back pain, coming on about an hour after food, with occasional vomiting of green watery fluid.

Nothing specific was apparent on clinical examination. Barium meal showed the typical semi-circular filling defect with stretched valvulæ conniventes of a small retrograde intussusception. The appearances were most marked in the prone position. In the erect position the intussusception could be produced and reduced at will by abdominal palpation over the stoma.

He was treated on a medical régime, and when seen three months later stated that his symptoms were much improved. It has not been possible to follow him up further.

Comment.—Chronic recurrent retrograde intussusception, easily reducible during barium meal examination.

Case 10.—This 52-year-old man had had a gastro-enterostomy twenty-six years previously for duodenal ulcer.

Fourteen years later he developed further peptic ulcer-type symptoms, and a barium meal at that time showed a jejunal ulcer close to the anastomosis.

He was put on a medical régime and his symptoms were much improved, but over the next sixteen years he had occasional attacks of upper abdominal pain, relieved by vomiting and by alkalis. However, these attacks became somewhat more persistent and he was seen in out-patients on account of them. Physical examination revealed nothing specific. A barium meal showed coarse gastric mucosa, with a well-defined filling defect above the stoma due to efferent loop herniation. The intussusception was readily reducible by palpation over the anastomosis. The duodenal cap was also noted to be grossly deformed. Medical treatment was instituted and he has not been seen again.

FIG. 291.—Same case five months later. Chronic recurrent intussusception. The filling defect is still present, but both afferent and efferent loops have now filled with barium.

Comment.—Chronic recurrent intussusception, present twenty-six years after the original gastro-enterostomy, which was seen to be reducible on the radiographic screen.

CLASSIFICATION

It is usual to divide retrograde jejuno-gastric intussusception into two main types: chronic recurrent and acute. It is suggested that in the interests of differentiation a third type, the acute post-operative, should be added, since it differs considerably from the ordinary acute type both in presentation and in clinical course.

Clinical Presentation.—

Acute Post-operative.—Typically, this presents on the fourth or fifth post-operative day in a patient who previously has been recovering normally. The patient develops colicky abdominal pain, accompanied by attacks of vomiting. The vomit is frequently bile-stained. These symptoms are often intermittent, and the patient may be free of them for 24 or 48 hours before they recur. Generally, the extent of the symptoms is not sufficient to give rise to grave anxiety. In only 1 out of 7 cases was it felt necessary to re-open the patient; in the other 6 cases of this type expectant treatment by gastric suction and intravenous fluid replacement resulted in gradual improvement and eventual cessation of the symptoms, without invoking further measures.

These symptoms are frequently difficult to distinguish from other causes of vomiting in the post-operative period, such as jejunal ileus or obstruction of the stoma due to malposition or leakage around it.

Nor are clinical signs of great value. Some upper abdominal distension may be present but beyond this no abnormality is usually apparent.

The diagnosis is likely to depend upon radiological demonstration of the lesion, or upon direct visualization at laparotomy.

Acute Delayed Type.—No example of this group is included in the present series. Descriptions in the literature of typical cases are to be found by Aleman (1948), Soteropoulos, Berkmen, and Gilmore (1959), and Paul and Benkendore (1959).

Typically, the patient has had a gastro-enterostomy some months or years earlier, and is then admitted to hospital with definite signs of an acute abdominal catastrophe. Colicky abdominal pain, bile-stained vomit, and occasionally hæmatemesis are the usual presenting symptoms. There are no characteristic signs, though a palpable upper abdominal tumour has been described. Plain radiographs of the abdomen may suggest obstruction at the stoma, but will not demonstrate the specific cause.

The condition of the patient is such as to suggest that immediate laparotomy is necessary, and hence barium-meal examination is only rarely performed pre-operatively. It is noteworthy that very frequently the history of these patients reveals occasional attacks of vomiting and colicky abdominal pain which resolve spontaneously prior to the final acute episode; moreover, the incidence of these attacks may extend back to the immediate post-operative period, suggesting that they may be examples of the chronic recurrent type, which on this occasion have failed to reduce spontaneously.

Chronic Recurrent Type.—Patients in this group may not present until many years after their original gastro-enterostomy. Thus in *Case* 10 the interval was twenty-six years. However, careful history-taking will often reveal a history of abdominal pain and intermittent vomiting which may extend over a considerable period of years. It is characteristic, but not invariable, for the pain to be relieved by vomiting. This may in fact have an anatomical basis, for considering the ease with which the intussusception may be produced and reduced under radiological observation, it seems reasonable to suppose that the increased intragastric pressure produced during the act of vomiting would be sufficient to effect spontaneous reduction of the intussusception. Although the history may be suggestive, it is not usually sufficient to differentiate this condition from other pathology at the stoma or from jejunal ulcer, and barium-meal examination is usually required to establish the diagnosis. The prolapsed loop has been visualized gastroscopically (Richardson, 1953).

Radiological Appearances.—In cases of acute delayed intussusception, plain films of the abdomen may reveal gaseous distension of the stomach and afferent loop suggestive of stomal obstruction, but not yielding specific information.

In a typical case, however, barium meal reveals a filling defect in the stomach above the expected site of the stoma, with its upper border convex in relation to the gastric fundus. The mucosal pattern of the jejunum is usually recognizable as such. It often manifests itself as a fan-shaped series of lines radiating outwards from the region of the stoma. These lines, of course, represent contrast which has been retained between the valvulæ conniventes. The appearances may not always, however, be as characteristic as this.

In some cases a semi-circular filling defect alone may be present, and the jejunal mucosal folds are not seen, presumably because the lumen of the intussuscepted segment has failed to fill. In two cases, the centre of the filling defect showed a small circular area of contrast, probably representing the jejunal lumen seen *en face* (*Figs.* 287, 290).

The afferent loop is often observed to be widened, with its mucosal folds stretched. At screen examination the contrast may be observed to fill the stomach, pass around the dilated afferent loop, up to the region of the stoma, but not pass into the efferent loop (*Fig.* 286).

Filling of the efferent loop is inconstant. In the acute post-operative type, the efferent loop usually does not fill, or only a very small quantity of contrast enters it, but in the chronic recurrent type, the efferent loop may fill readily and there will be little or no delay at the stoma. Apart from this, the radiological appearances in the acute and chronic types are essentially the same, except in so far as the size of the intussuscepted segment is frequently smaller in the chronic group.

Furthermore, in this type, the intussusception may readily be produced and reduced on the screen by abdominal palpation over the stoma, whereas in the acute type palpation does not alter the appearances.

It is important that the patient should be examined in the supine and, if possible, in the prone position, since the effect of gravity in the erect position may be sufficient to obscure the herniated segment.

Aleman (1948), in reviewing approximately 70 cases reported up till that time, was able to assess the anatomical derangement in 58. He found the distribution to be as follows:—

Type I, Afferent loop intussusception	6
Type II, Efferent loop intussusception	43
Type III, Both loops intussuscepted	9

These figures were based partly on operative, and partly on radiological findings.

The experience of the present writers is that this differentiation into types is exceedingly difficult on radiological grounds alone, nor does it appear to be of any great clinical significance, nor to be likely to influence the treatment. Thus, even if the afferent loop alone is herniated, this may in itself be sufficient to obstruct the outflow from the efferent loop, and the resultant mechanical effect will be the same as if the efferent loop itself were intussuscepted.

TREATMENT

In 7 cases of acute post-operative intussusception here discussed, operative treatment was necessary in only 1. In this case, symptoms were manifest on the third post-operative day, and laparotomy was carried out on the following day. No abnormality was found at the stoma. Negative operative findings have been recorded before, and it is probable that traction on the mesentery, whilst locating the stoma, is sufficient in itself to reduce the intussusception.

In the other 6 cases, recovery followed a régime of intragastric suction and intravenous fluid. It seems likely that, in the absence of any definite contra-indication, this should be the first line of treatment.

It is noted that in all 6 cases clinical improvement followed within a few hours of barium-meal examination. This occurred sufficiently frequently to suggest that it is more than mere coincidence. Possibly the actual weight of barium, coupled with the gastric peristalsis that its presence induces, may be sufficient to reduce an intussusception which is not yet incarcerated. There is no report of spontaneous reduction of a devitalized segment of bowel.

In the acute delayed type urgent surgical intervention is usually required, since it is in these cases that gangrene of the bowel is likely to occur. It is reported that if operation is delayed more than 48 hours the mortality rises to over 50 per cent.

In the chronic recurrent type the symptoms may not be severe enough to justify any surgical procedure, and medical treatment may be sufficient. It has been suggested that large doses of sodium bicarbonate may maintain sufficient pressure within the stomach to prevent jejunal prolapse occurring, but its success has not been a significant feature in the 1 patient in this series submitted to this régime.

In *Case* 8 in this series, in whom the symptoms were persistent, the anastomosis was undone and a Billroth-I gastrectomy performed, thus restoring the normal circuit. It appears that this is a satisfactory procedure, if it is practicable in any individual case.

DISCUSSION

The cause of retrograde jejunogastric intussusception is not known. It differs from intussusception at other sites in adults, where some definitive cause, such as a tumour, is frequently present at the apex of the intussuscepted segment. Amberger (1921) found a fibroma associated with a jejunal intussusception, but this case appears unique, and no such cause is usually apparent.

It has been suggested that reversed peristalsis in the jejunal loop may be responsible for causing the herniation through the stoma, but this would be impossible to prove. It is certainly not apparent at fluoroscopic examination.

Three of the 7 patients with post-operative intussusception had had vagotomy in association with a gastro-enterostomy. In these circumstances the stomach is almost invariably aperistaltic (Jackman, 1961), whereas the peristalsis in the jejunal loop is normal or increased. In this instance, the purely mechanical factors would be such as to tend to force the jejunum through the stoma, but such an explanation can only be speculative.

Two main conclusions appear to emerge from this study. First, as in *Case* 7, acute symptoms may arise in the post-operative period, subsequently subside, then recur in the typical chronic recurrent form.

Moreover, reports in the literature of the acute delayed type indicate that not infrequently the patient has had sub-acute symptoms dating from the immediate post-operative period, before presenting as an acute upper abdominal catastrophe. This suggests that minor prolapses may have occurred, which have reduced spontaneously, prior to the final acute attack.

Secondly, it is suggested that retrograde intussusception is in fact more common, particularly in the post-operative period, than is generally believed.

Since the majority of cases undergo spontaneous reduction the diagnosis may easily be overlooked. This is particularly true in that group of patients who develop vomiting in the post-operative period, but who respond to routine treatment with gastric suction and intravenous fluids. Their symptoms are frequently ascribed to jejunal ileus, or stomal œdema, without the cause being accurately determined. Barium meal may be omitted, perhaps because the patient is not considered well enough to be transferred to the X-ray department. It has been shown that in such cases an intussusception may be demonstrated or excluded by administering 2 oz. of neat barium in the ward, and taking two abdominal pictures with the ward mobile apparatus, with minimal disturbance to the patient.

It is also possibly true that in a number of cases the barium meal appearances have been misinterpreted both by the radiologist and by the clinician, due to relative unfamiliarity with the appearances.

A more thorough search for the condition in the appropriate circumstances may lead to its more frequent recognition.

SUMMARY

Ten cases of retrograde jejunogastric intussusception, with radiological confirmation, are discussed. It is shown that they fall into three clearly defined groups: acute post-operative, acute delayed, and chronic recurrent. Further, it is suggested that in both the acute delayed and in the chronic recurrent forms the symptoms have frequently been present, although less severe, since the time of the original operation. This indicates that the three various types are manifestations of essentially the same abnormal process, although the clinical presentation may be very different.

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ABDOMINAL ACTINOMYCOSIS, WITH SPECIAL REFERENCE TO THE STOMACH

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ACTINOMYCOSIS affecting the abdominal organs appears to be a rare disease. In forty years of hospital practice I have notes of only 16 patients with this disease; 14 of these have occurred during the last 36 years, during an association with hospitals which now average over 6000 surgical admissions yearly. Since 1948, when a disease index was begun, only 1 other patient has been traced. This may not represent the true incidence of the disease, but it gives some indication of its rarity.

Cervicofacial actinomycosis is more common. My dental colleague, Mr. Paul Bramley (Bramley and Orton, 1960), has diagnosed 11 cervicofacial cases in two years.

My interest in actinomycosis was aroused in student days when I was able to observe a patient who was operated on for gangrenous appendicitis, followed by infection of the abdominal wall, and spread of the disease to the liver and through the diaphragm to the lung, and finally to see the complete pathological picture at the autopsy.

At about the same time, in routine section-cutting from post-mortems, a small white nodule from the centre of the liver of a child aged six who had died immediately after a 'run over' accident, surprisingly proved to be a clump of actinomycoses. No clue of its origin had been shown in the careful autopsy demonstration.

Table I sets out the age, sex, proof of diagnosis, treatment, and result of the 14 cases.

The disease appears more often in association with appendicitis, but in other organs the symptoms and signs can assume bizarre forms. In this series, 8 cases were associated with appendicitis, and followed the classical description, and 6 cases were unusual in location or presentation. Because of its rarity the diagnosis is often not made until an advanced stage has been reached, with corresponding difficulty in treatment. Cope (1949) calls it "the most misdiagnosed disease", and there is "no disease which is so often missed by experienced clinicians".

When an abdominal condition not conforming to the usual clinical picture is encountered, awareness of the possibility of this disease should lead to further consideration of the physical signs, and, if possible, bacteriological confirmation.

The classical physical sign is a wooden hard mass. In the early stages this area of hardness, although extensive, may not show any reddening of the overlying skin and is not very painful or tender. It can be observed to increase slowly in size with involvement of the skin and the production of indolent sinuses which, for the size of the indurated area, discharge a relatively small amount of pus. The patient suffers a steady decline in health with a low-grade fever, and occasional bouts of higher fever and local discomfort. Clinical suspicion is often not aroused unless sulphur granules are seen, leading to a specific request for search for actinomyces. Routine Gram films of pus may show organisms morphologically resembling *Actinomyces* even in the absence of macroscopic granules, and this finding will result in the appropriate anaerobic culture. The possibility of successful identification and culture is improved if a quantity of pus, rather than a dried-up film on a swab, is available for examination. It should be noted that unless the pus is received directly into a test-tube, with minimum exposure to air, and quickly taken to the laboratory, culture may be unsuccessful. In Bramley and Orton's cases (1960), this procedure produced cultures which were tested for sensitivity to various antibiotics.

If, under the mistaken impression that a quantity of pus is present, the mass is incised, the peculiar hardness and the presence of small areas of suppuration make suspicion almost a certainty.

There are some abscesses which, owing to the other organisms present, will produce a large quantity of pus and may appear to be simply pyogenic but fail to heal after adequate drainage, and therefore repeated examination of pus is necessary, a point which is easily forgotten when reports state that numerous organisms sensitive to one or other antibiotic are present.

Failure to diagnose the disease will lead to inexorable extension, and this delay may, even with potent antibiotics, lead to fatality. The clinical signs are of paramount importance and it is the knowledge of these that triggers off in the mind the thought "this may be actinomycosis". If the clinical picture indicates this disease, treatment should begin without waiting for bacteriological confirmation.

CASE REPORTS

(*see Table I*)

Cases associated with Appendicitis.—

Cases 11 and 12 have some interest, as a hard mass appeared at or near the operation site, following unperforated appendicitis, and responded rapidly to large doses of penicillin, without the formation of an abscess, which usually occurs despite antibiotics if the infection is due to the usual suppurative organisms. In *Case* 13 the condition was recognized by the operator (Mr. M. Solan); the appendix was not removed, and penicillin produced a rapid cure. *Case* 14 illustrates the difficulty of cure when diagnosis is delayed.

Case 14.—Male, aged 14 years. He had been treated elsewhere, with the following history:—

March, 1960.—Fourteen days' abdominal pain, mass in the right iliac fossa. Attempted appendicectomy, but the firm adhesions prevented removal of the appendix. One month later an abscess in the incision was drained.

May, 1960.—Appendix removed, showing subacute inflammatory reaction and a small amount of pus between the adherent areas of the ileum and

ascending colon. Crystamycin was given, but after twelve days the wound discharged pus. Chloramphenicol produced some improvement, but in July a sinus persisted with a hard non-tender lump, approximately 3 in. in diameter, to the left of the paramedian incision.

An occasional Gram-positive filament resembling *Actinomyces* was eventually found after examination of many specimens of pus.

July 29.—Penicillin 500,000 units b.d., and on Aug. 9 increased to 1 mg. b.d.

Aug. 15.—Progress was slow and Lugol's iodine 5 min. t.d.s. and sulphadiazine 1 g. t.d.s. were added.

Aug. 23.—Gram-positive filamentous organisms showing apparent branching and morphologically consistent with *Actinomyces*.

Aug. 25.—Penicillin 3 megaunits, daily.

Sept. 4.—Discharged, to continue penicillin, etc., but refused further injections. Tab. broxil was given, but when seen on Oct. 3 he had lost weight and a small fæcal fistula was present.

As it had not been possible to cultivate the organism, the sensitivity was unknown, and on the assumption that it was now insensitive to penicillin, tetracycline (ledermycin) 250 mg. 6-hourly was ordered, with Lugol's iodine 5 min. t.d.s.

Nov. 15.—This treatment seemed ineffective and, the discharge from the fistula, which was now certainly from the small intestine, had increased. A sinogram, however, showed that the opaque medium entered the descending colon. Erythromycin 500 mg. q.d.s. was begun. This treatment appeared effective as the induration lessened and it seemed that closure of the fistula was possible.

Jan. 1, 1961.—The fistula was dissected out with difficulty owing to the dense fibrosis. Three inches of ileum, which were very indurated, were resected, and adhesions of ileum to descending colon separated, but no fistulous communication with the colon was found. Following this, the incision suppurated and a fæcal discharge began.

The appetite improved at once, and weight was regained. The suppuration and fæcal discharge gradually diminished and he was fit for discharge at the end of February, but the fæcal fistula failed to heal. His health improved and operation was postponed as long as possible owing to his previous long stay in hospital. At re-operation (Aug. 11), after dissection through hard fibrous tissue, the ileum was found adherent to the descending colon and considerably stenosed. Six inches of ileum, containing two fistulæ, and the cæcum, were resected; continuity was restored by side-to-side anastomosis. A small fistula in the descending colon was oversewn.

Table I.—Cases of Abdominal Actinomycosis Collected over 36 Years

No.	Age	Sex	Organ	Year	Diagnostic Proof	Treatment	Result
1	21	F	Appendix	1926	Bacteriology	Iodine	Died
2	40	F	Appendix	1926	Bacteriology	Iodine	Cure
3	45	F	Appendix	1927	Bacteriology	Iodine	Cure
4	25	F	Appendix	1939	Bacteriology	Iodine, radiotherapy, sulphanilamide	Cure
5	40	M	Appendix	1940	Bacteriology		Died
6	32	M	Appendix	1944	Bacteriology	Iodine, radiotherapy, penicillin	Cure
7	75	M	Appendix	1950	Bacteriology	Penicillin, sulphadiazine, iodine	Cure
8	61	M	? Renal	1951	Post-mortem	Antibiotics	Died
9	53	F	Stomach	1954	Bacteriology	Penicillin	Cure
10	16	M	Appendix	1955	Bacteriology	Antibiotics	Cure
11	19	M	Appendix	1956	Clinical only	Penicillin	Cure
12	49	F	Appendix	1958	Clinical only	Penicillin	Cure
13	10	F	Appendix	1960	Clinical only	Penicillin	Cure
14	14	M	Appendix	1960	Bacteriology	Penicillin, iodine, sulphadiazine, erythromycin	Cure

Recovery was normal and the patient is now well. Histological examination showed a simple granulomatous reaction in the cæcum and isolated areas in the small bowel. Plasma cells and eosinophils predominate in all lesions. Histological evidence of active actinomycosis not found.

Actinomycosis of Stomach.—

Case 9.—Female, aged 53 years.

Admitted July 13, 1954, as an emergency, with epigastric pain after an attack of vomiting three days before. The pain had moved to the left hypochondrium. Temperature 100·2°; pulse 108; slightly jaundiced—serum bilirubin 1·2 mg. 100 ml.; direct van den Bergh, weak positive. Some congestive signs in the lower lobe of the right lung were present, with tenderness in the right hypochondrium, with some guarding and possibly a palpable mass, and some tenderness in the left hypochondrium also. A diagnosis of acute cholecystitis was made and sulphadiazine prescribed. Radiography of the chest showed some consolidation in the right base; no shadows in the gall-bladder area.

Operative treatment was deferred because of the state of the lung. Sulphadiazine was continued for eleven days; the temperature had fallen, pain diminished, and the lung condition had improved. She was discharged fourteen days after admission, to return later for cholecystectomy.

Sept. 19.—Re-admitted. She had lost 2 stone in weight, attributed to dieting. There had been no pain but some soreness in the right hypochondrium. A hard tender mass could now be felt immediately

above the umbilicus, occupying one-third of the epigastrium. Temperature 98·4 ; pulse 100.

Sept. 23.—Opaque meal. "Well-marked indentation in the region of the pyloric part of the stomach, caused by pressure from a hard fixed mass, extrinsic to stomach and could well represent a pancreatic lesion."

Sept. 29.—On my return from holiday I thought the mass was probably a pancreatic cyst and requested further radiographic examination.

Oct. 13.—Opaque meal. "The defect of the gastric antrum is much more pronounced than at the previous examination. There is a definite half-shadow at the proximal end of the lesion and the extension through the antrum itself appears to be integral with the gastric wall. The pylorus is fairly free and the duodenal cap fills out. The pattern of the duodenum crossing to the duodeno-jejunal flexure does not suggest a pancreatic lesion, at any rate of the inflammatory type. The appearance is that of an invading neoplasm of the stomach wall, possibly from an extrinsic source, but the defect is completely unlike a simple pressure, nor does it suggest involvement of the pancreas." (G. Fildes.) *See Fig.* 292. As a check on the possibility of a gastric neoplasm, a fractional test meal showed "Nearly total achlorhydria. No blood in resting juice." The temperature during three weeks ranged from 97·4° to 99°.

FIG. 292.—Oct. 13, 1954.

Oct. 18.—There was now a fluctuant area, slightly reddened, 1 in. above the umbilicus. The diagnosis now appeared to be a pancreatic abscess, and on Oct. 20 the fluctuant area was incised. A small amount of brown slimy pus was found, and a drain inserted.

Bacteriology of pus: "A moderate number of pus cells, and Gram-positive filamentous organisms showing branching and 'granule' formation. The morphological appearances are those of a member of the Actinomycetes group. Aerobic and anaerobic culture—no growth after 24 hours' incubation." Further culture grew a mixed growth of anaerobic streptococci and organisms of the Bacteroides group.

Oct. 25.—Penicillin 500,000 units b.d. and sulphadiazine 1 g. four-hourly ordered on the assumption that the disease was actinomycosis.

Nov. 3.—Opaque meal (for comparison). "The antral defect is still present but is less marked than formerly and the normal gastric rugæ are visible. In the prone position there was some motility visible in the area. Appearances suggest regression of the disease." (*Fig.* 293.) The treatment was continued until discharge on Nov. 7. One million units of long-acting penicillin was given daily for fourteen days as an out-patient. A total of 43 megaunits penicillin and 52 g. of sulphadiazine was given.

FIG. 293.—Dec. 2, 1954, after 8 megaunits of penicillin.

Dec. 4.—Opaque meal. "The deformity previously noted in the pyloric antrum is now much less marked, suggesting that the condition is improving." Her condition steadily improved, and when seen in March, 1955, she seemed quite well and an opaque meal was reported as follows: "The stomach and duodenum now show absolutely normal appearances." (G. Fildes, March 10, 1955.) She was re-examined in January, 1961, and was in excellent health.

Comment.—The administration of sulphadiazine during the first stage of the disease was sufficient to lead to a temporary remission of symptoms. When the swelling was found on re-admission my mental processes in diagnosis were directed towards the pancreas as I had been convinced that the original attack was acute cholecystitis. For that reason radiological suggestion of neoplasm was unacceptable, and the absence of blood in the resting juice favoured

withholding exploration. This was fortunate as a wide exploration might have produced a disaster.

Shanks and Kerley (1950) state that actinomycosis of the stomach is extremely rare and has been diagnosed as carcinoma, which is not surprising considering the macroscopic pathology which produces rigidity of the gastric wall, with irregularity and diminution of the gastric capacity, the usual radiological signs of carcinoma.

CASES FROM THE LITERATURE

Shearburn (1943) reports 2 cases.

1. Female, aged 24 years, who had recurrent epigastric pain for six or seven years. No radiographic examination is mentioned. At operation a mass adherent to the head of the pancreas obstructing the pylorus was found. Biopsy of the stomach showed clubbed mycelia. Gastro-enterostomy was performed, no other treatment given, and the patient was reported as being asymptomatic after eighteen months.

2. Male, aged 41 years. Perforated duodenal ulcer, Oct., 1940. Subphrenic abscess, July, 1941, followed by multiple abscesses. *Actinomyces* found in pus, Oct., 1941. Treated by thymol and a course of sulphadiazine. More abscesses were incised at different dates, and another course of sulphadiazine with improvement, but it appears that the patient probably died in April, 1943.

Raper (1950) reports a case of abdominal actinomycosis following perforation of a duodenal ulcer in a female aged 25 years. Numerous abscesses developed, including a possible lung abscess, over a period of two and a half years before *Actinomyces* were found. In three weeks, 10·5 megaunits of penicillin were given with satisfactory results, but three months later further treatment by 96 megaunits of penicillin in fifty days, in addition to iodine, was needed before cure was obtained.

Till (1946) reports a case following perforation of a gastric ulcer, where the diagnosis was made after four months. Treatment by iodine was apparently successful, but two years later the disease reappeared, and, despite further treatment by iodine, death occurred about four years after the onset of the disease.

It should be noted that this illness began in 1934 and terminated before sulphonamides were in use.

Schinz and Baensch (1928) state that *Actinomyces* invade gastric ulcers and tend to spread towards the greater curvature and invade the mesocolon and transverse colon, and finally infiltrate the abdominal wall. The infiltration of the gastric muscle causes filling defects in a radiograph—usually a notched filling defect. This appearance would lead to a diagnosis of carcinoma unless infiltration of the abdominal wall, together with a sinus, suggests another diagnosis.

Hitzenberger observed a duodenal fistula, due to actinomycosis, in which barium passed from the duodenum to a sinus in the right axilla.

Hadjipetros (1920).—Male, aged 54 years. Tumour in left hypochondrium, as large as a fist. Resection of part of the stomach and transverse colon. The mass was between the stomach and the colon and the mucosal surface of the stomach showed a radiating scar, and in another area there was the appearance of a healed ulcer. Numerous small abscesses were present in the mass, and typical *Actinomyces* were found. One year after operation the patient was well.

Israel (1889).—Female, aged 37 years, whose illness commenced apparently as peritonitis in January, 1888. After ten days a mass appeared and later disappeared. In May and June further attacks followed, leading to hospital admission, when a lump was found above and to the right of the umbilicus attached to the abdominal wall. After three weeks an abscess formed and in this *Actinomyces* were found. In August the mass was incised, curetted, and tinct. iodi injected into the mass on five occasions. In October the patient was apparently cured.

Prutz (1912).—Male, aged 36 years, with a huge mass in the left side of the abdomen in which *Actinomyces* were found. The evidence that this originated in the stomach is slender.

Pohl (1912).—Male, aged 36 years. Two months' illness, loss of weight, fever, and a mass in the right hypochondrium. At operation a mass of granulation tissue was found behind the stomach, which was adherent to the undersurface of the liver, and, by palpation through the anterior gastric wall, a large ulcer was felt. *Actinomyces* were found in the pus, potassium iodide was given for two months, and the patient was apparently cured.

Fuller and Wood (1945) record a male aged 48 years with a five-year history of digestive trouble. Radiographic examination showed a penetrating ulcer of the mid-portion of the lesser curvature. Three and a half months of medical treatment was unsuccessful, and at operation there were tumour masses on the lesser curvature. Partial gastrectomy was performed. The specimen was a disk-like mass 5 cm. in diameter and up to 2·5 cm. in thickness; microscopy showed abscesses filled with purulent exudate containing small colonies of ray fungus. No specific treatment for actinomycosis was given and the patient was in good health after fifteen months.

Blain (1933) records a male aged 37 years, whose radiograph showed a deformity of the stomach producing obstruction, diagnosed as chronic gastric ulcer. At operation a mass was found in the lesser curvature and a very radical subtotal gastrectomy performed. The specimen showed several abscess cavities. Three months later a subphrenic abscess was suspected but nodules in the liver were found. At autopsy five months later actinomycotic abscesses of the liver were found, and re-examination of the stomach showed the ray fungus. Blain quotes Nathan (1929) as stating that only 1 of 6 reported cases stands strict scrutiny.

Nathan (1929).—Male, aged 46 years. Two months' epigastric pain, melæna, and hæmatemesis. Radiograph showed perforation of gastric ulcer into liver. Wassermann +++. P.M., gastric ulcer penetrating liver. Ulcer on lesser curve 6·5 cm. × 14 cm. adherent also to the pancreas and duodenum. Abscess in right lobe of liver, and small pulmonary abscesses. *Actinomyces* were present in the ulcer and liver.

Behring (1933).—Female, aged 67 years. Six months' intermittent digestive symptoms with loss of weight. Radiograph showed pyloric stenosis. Operation: Firm tumour in pyloric area with adherent gall-bladder containing a small stone. Resection of first

part of duodenum and pyloric end of stomach, Billroth I anastomosis. The patient was well two months later.

Specimen: No ulcer or cicatrix, but duodenal wall thickened, and on the posterior wall an abscess cavity 3 mm. in diameter opening into the lumen of the duodenum by a track 3 mm. long, with extension elsewhere under the mucosa. These changes are sharply limited by the pyloric ring. Behring states that the portal of entry can seldom be demonstrated as it is minute, and therefore the mycelia escape the bactericidal action of hydrochloric acid.

TREATMENT

In the present-day treatment of peritoneal infection, the almost routine administration of powerful antibiotics is likely to lead to the masking of some cases of actinomycosis. The dosage will be sufficient to tide the patient over the emergency and recover well enough for discharge from hospital, but, on cessation of treatment, the remaining fungi may grow and produce some manifestation of disease which may be remote from the original lesion.

In the past iodine or iodide has been successful, probably where the disease has been of limited extent. It possibly has still some value for its effect on the fibrous tissue. Providing iodism does not occur, it may be used for long periods in small doses of the tincture as an adjunct to other treatment.

The sulphonamides are valuable but the risks of toxic effects when given for a long period are present. Some patients will tolerate sulphadiazine 0·5 g. thrice daily for many weeks. *Case* 9 received 52 g. of sulphadiazine, and this appeared to hasten the cure.

The use of radiotherapy is open to question. It is possible that it has some effect in softening the fibrous tissue but the possibility of remote effects makes its employment potentially dangerous.

Surgery, apart from the initial operation for the abdominal emergency, has no place other than the evacuation of definite abscesses, but it should be noted that the cases reported by Fuller and Wood (1945), Hadjipetros (1920), and Behring (1933) recovered after partial gastrectomy, and that of Shearburn (1943) after gastro-enterostomy, without specific treatment. It is difficult to explain this, as Blain's case (1933) died after similar treatment.

The availability of penicillin has changed the picture remarkably. From the limited experience of this group of cases, it would seem that penicillin in doses of 2 megaunits daily, given until all signs of induration have disappeared, has been the most successful treatment. The total amount given has varied and it is not possible to specify how much is needed. It is probably wise to continue the treatment for one, or possibly two, months after all physical signs have disappeared, as it is not clinically possible to be certain that every focus has been eradicated. This appears to be borne out by some of the recorded cases where, after apparent cure, the disease reappeared in another site many months later. The organism is sensitive *in vitro* to other antibiotics, but owing to their side-effects it may not be possible to use them for a sufficient time to ensure cure. In several cases of this series various wide-spectrum antibiotics have been given for short periods with little apparent effect on the course of the disease.

Treatment of anæmia by iron or even transfusion is necessary, and nursing under ideal conditions will hasten recovery.

SUMMARY

1. Fourteen cases of abdominal actinomycosis are described, including 1 involving the stomach.

2. The clinical picture and its influence on diagnosis is discussed.

3. Present-day treatment of peritoneal suppuration, and the possible pitfalls influencing the diagnosis, are considered.

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I have learnt much from the writings of Sir Zachary Cope who has done so much to remind the profession of the nature and existence of this disease.

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AORTIC EROSION COMPLICATING ŒSOPHAGO-GASTRECTOMY

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THE mortality from the operation of œsophago-gastrectomy for carcinoma is high. If the broadest interpretation is given to operative mortality to include all patients dying from a pathological process initiated by operation, whether or not these patients die in hospital, and without reference to the time interval between operation and death, then, in a series of 418 resections for œsophageal carcinoma reported elsewhere (le Roux, 1961), the operative mortality is 30 per cent. One of the late complications of œsophago-gastrectomy is massive fatal hæmorrhage from erosion of the aorta, the aortic fistula usually communicating with the alimentary tract through a defect in an œsophago-gastric anastomosis. Ten examples of this complication are described, accounting for a little less than 8 per cent of the operative mortality in the series to which reference has previously been made. The pathological processes which seem likely to precede the hæmorrhage are discussed, a recognizably consistent clinical picture is drawn, and surgical management which may avert death is outlined.

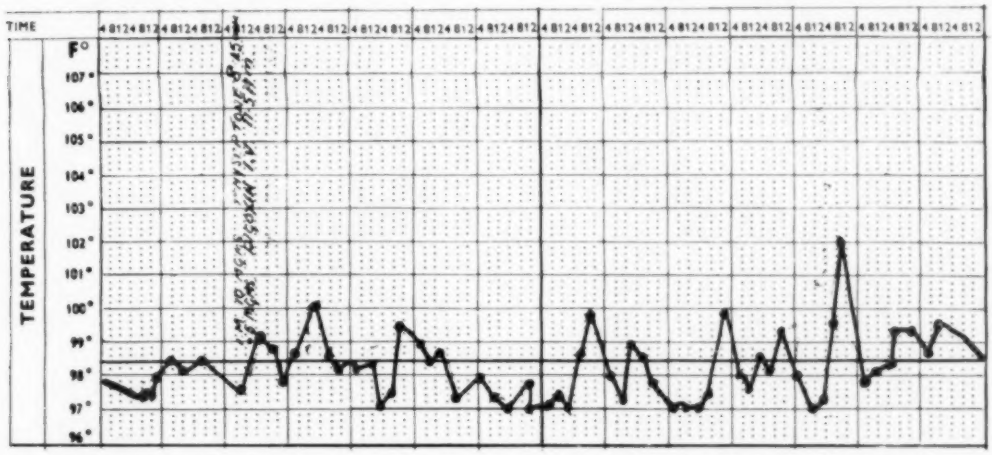

FIG. 294.—Temperature chart from *Case* 1 showing a rise in temperature almost every day during the first fortnight after operation.

CASE REPORTS

Case 1.—A 77-year-old man with dysphagia for 2 months was shown to have an adenocarcinoma at the cardia, 41 cm. from the upper alveolus. Œsophago-gastrectomy was undertaken in January, 1953. The distal part of the stomach was used to replace the œsophagus, and an end-to-end œsophago-gastrostomy was fashioned with interrupted silk sutures at the lower border of the aortic arch. An early complication was auricular fibrillation, recognized on the second post-operative day. Rate and rhythm had been restored to normal with digoxin by the fifth post-operative day. From the third post-operative day the patient was pyrexial at some time each day, the fever rising to 99–100° F. (*Fig.* 294). He was ambulant on the fifth post-operative day, and thereafter developed a little ankle œdema; fever and ankle œdema did not persist after the fifteenth post-operative day and he was discharged on the twenty-third day. On the twenty-seventh day, at home, he complained of severe retrosternal pain, and felt weak. Normally hypertensive, he was found by his general practitioner to be hypotensive and was admitted to a local hospital. On the twenty-eighth day he had melæna. On the morning of the twenty-ninth post-operative day he vomited a small quantity of bright-red blood. His pressure again fell and he was given a transfusion of blood. That evening he died from massive hæmatemesis.

At autopsy there was an abscess cavity the size of a golf-ball in the mediastinum, lying between the œsophago-gastrostomy and the aorta, and where the aorta formed the wall of the abscess cavity its adventitia was necrotic and there was a perforation into the aortic lumen which would admit a probe. There was a defect in the œsophago-gastric stoma where this formed part of the abscess wall, and this defect would admit a lead pencil. In the abscess cavity, and in the lumen of the œsophagus and stomach, there was fresh blood-clot.

Case 2.—A 46-year-old man who had had dysphagia for 2 months was shown to have an adenocarcinoma of the œsophagus 35 cm. from the upper alveolus. Œsophago-gastrectomy was undertaken in November, 1957. Glands about the cœliac axis and in the tracheal bifurcation contained tumour. End-to-end œsophago-gastrostomy was made with interrupted silk at the lower border of the aortic arch. On the fifth post-operative day, having been ambulant for 2 days, the patient was found to have a painful swollen right calf. He was confined to bed, the foot of which was elevated, and he was given heparin 10,000 units four times a day for 2 days, then 7,500 units four times a day for 2 days, then 5,000 units four times a day for 2 days, and the same dose three times a day for a further 4 days. His temperature rose to 100° F. with the development of venous thrombosis, and fever persisted despite the rapid return of the swollen calf to normal. On the fifteenth post-operative day—the last day of the

course of heparin, he vomited a little blood. He was given protamine sulphate and a transfusion of blood. Five hours later he died from massive hæmatemesis.

The findings at autopsy were little different from those described in *Case* 1. The defect in the œsophago-gastrostomy was smaller, admitting only a probe; the abscess cavity was smaller—the size of a cherry; and the defect in the aorta was accurately placed 5 cm. from the origin of the left subclavian artery on the anteromedial aspect of the aorta and admitted a small probe.

FIG. 295.—In the lower half of the photograph the arrow is emerging through the defect in the œsophago-gastrostomy, the mucosal aspect of which is seen. In the upper half of the photograph the erosion in the aorta is seen from within the aortic lumen.

Case 3.—A 49-year-old man with increasing dysphagia for 3 months was shown to have an adenocarcinoma of the œsophagus 37 cm. from the upper alveolus. Œsophago-gastrectomy was undertaken in June, 1959, the œsophago-gastric stoma lying at the lower border of the aortic arch. He was pyrexial between the second and tenth post-operative days, his chart being similar to that shown in *Fig.* 294. He was ambulant from the second post-operative day, ate a normal diet from the ninth day, and was discharged on the fourteenth day. On the evening of discharge he had a small meal at home and went to bed, feeling very tired; he woke at midnight coughing and spluttering and found large clots and bright red liquid blood on his pillow. He was re-admitted a few hours later and a transfusion of blood was given. Preparations were made for thoracotomy, and while awaiting anæsthesia, eight hours after the initial episode of hæmorrhage, he vomited a large quantity of blood and became pulseless. The trachea was intubated and the left chest was opened. There was no cardiac action, but with the rapid intra-aortic transfusion of three pints of blood, and massage during the transfusion, the heart started. Before the transfusion was begun the aorta was cross-clamped immediately distal to the left subclavian artery and a second clamp was applied close to the diaphragm. The descending thoracic aorta was stripped from the intrathoracic stomach and the proximal aortic clamp momentarily released. The site of aortic erosion was immediately seen and the clamp re-applied. The ventricles then fibrillated. Massage and defibrillation did not restore cardiac action to normal. Massage, although it produced a palpable carotid pulse most of the time, was apparently inadequate because the pupils

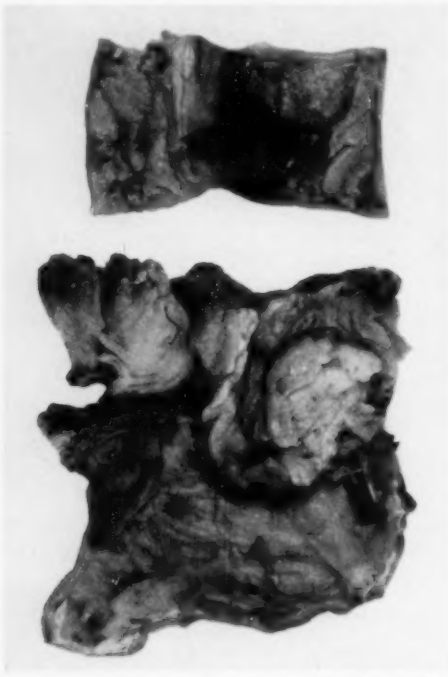

FIG. 296.—In the upper half of the photograph the black slough on the adventitial aspect of the aorta can be seen with the arrow emerging through the erosion. In the lower half of the photograph the œsophago-gastrostomy is seen from the external aspect with the site of leakage from the anastomosis indicated.

dilated. At no time had the patient received any anæsthetic drug. When the aorta had been cross-clamped for an hour, and the pupils had remained fixed and dilated for 30 minutes, attempts at resuscitation were abandoned. Further examination of the aorta showed the erosion to lie 8 cm. distal to the origin of the left subclavian, and 4 cm. distal to the œsophago-gastrostomy. The œsophagus and stomach were full of clot, there was a defect in the stoma which would admit a lead pencil, and black slough covered an area 1 cm. in diameter on the anteromedial aspect of the aorta, with a perforation admitting a probe in the middle of the slough (*Figs.* 295, 296). The action of stripping the stomach from the aorta had destroyed the evidence of a communication between the aorta and the œsophago-gastrostomy. There was nothing to suggest an abscess cavity with the stomal and aortic defects opening into it. At the most, there was a narrow track joining the two defects.

Case 4.—A 61-year-old man with dysphagia for 3 months was shown to have an adenocarcinoma 38 cm. from the upper alveolus. Œsophago-gastrectomy was undertaken in May, 1960. Mediastinal glands were

invaded by tumour. Apart from intermittent fever to 99·6 F. for 12 days, convalescence was uneventful. He was discharged on the seventeenth post-operative day able to swallow a normal diet without difficulty. Three and a half weeks later—in the sixth post-operative week, he complained of feeling generally unwell and lacking appetite. The day after the onset of these symptoms he vomited a small quantity of blood. He was admitted to a local hospital and was observed for 2 days, during which he was apparently well. He was found dead in bed during the early hours of the morning of his third day in this hospital. There was a large volume of blood and clot in the bed. The autopsy report is of direct communication between aorta and œsophago-gastrostomy through a probe-sized hole, without intervening abscess.

treated by transfusion and sedation. He died 30 hours later from massive hæmorrhage. At autopsy there was an abscess cavity in the mediastinum a little larger than a golf-ball, walled by aorta, left main bronchus, and the œsophago-jejunostomy. More than half the circumference of the œsophago-jejunostomy had disrupted. Bleeding had occurred from a right intercostal artery traversing the wall of the abscess. The abscess cavity and the proximal alimentary tract were filled with blood-clot.

Case 7.—An unusually small 73-year-old female who had had dysphagia for 4 months was shown to have a squamous carcinoma of the œsophagus 24 cm. from the upper alveolus. Œsophago-gastrectomy was undertaken in May, 1956. The distal part of the stomach was used to replace the œsophagus and although the anastomosis was

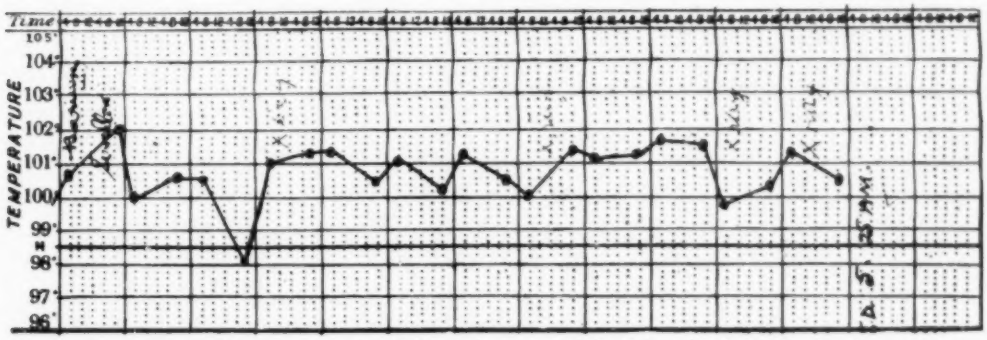

FIG. 297.—The temperature chart during the last 12 days of life of the patient described as *Case* 6.

Case 5.—A 68-year-old woman who had had dysphagia for 3 months was shown to have an undifferentiated carcinoma of the œsophagus 28 cm. from the upper alveolus. Œsophago-gastrectomy was undertaken in October, 1959. The cardiac end of the residual stomach was closed and a supra-aortic anastomosis was made between the gastric fundus and the œsophagus, the stomach lying lateral to the aortic arch. Convalescence was relatively uneventful, but she was pyrexial, with a temperature of 99·6 F. most of the time. On the morning of the fourteenth post-operative day she complained of feeling faint, and an hour later vomited a small quantity of blood. Two hours later she had melæna. A blood transfusion and a sedative were given. Five hours after the initial hæmorrhage she died from massive hæmatemesis. At autopsy there was a fistulous communication between the aorta 6 cm. distal to left subclavian and the site of closure of the cardiac end of the stomach. The stomach had been closed in three layers—catgut for the mucosa, and then two layers of interrupted silk. The gastric closure lay against the lateral aspect of the descending thoracic aorta, and there was no intervening abscess cavity.

Case 6.—A 54-year-old man who had had dysphagia for 3 months was shown to have a squamous carcinoma of the œsophagus 29 cm. from the upper alveolus. Œsophago-gastrectomy was undertaken in October, 1954. Jejunum was used to replace the œsophagus and an end-to-end œsophago-jejunostomy was fashioned at the lower border of the aortic arch. From the second post-operative day the patient was pyrexial, with fever rarely below 100 F. and often more than 101 F. The chart of the second half of his convalescence is shown in *Fig.* 297. At 11 o'clock on the evening of the nineteenth post-operative day he vomited bright red blood. Four days before this he had developed auricular fibrillation and although the rate had been controlled with digoxin the rhythm had not been restored to normal. The initial hæmatemesis was made at the lower border of the aortic arch it retracted medial to the aorta and lay finally just above the aortic arch. Convalescence was entirely uneventful. At no time was she pyrexial. She became ambulant early, and 3 weeks after resection was transferred to a medical ward for further convalescence. She died there from massive hæmorrhage on the twenty-seventh post-operative day without any warning hæmorrhage. At post-mortem examination there was an abscess in the mediastinum about 1 cm. in diameter walled by left main bronchus, aorta, and the œsophago-gastric anastomosis. It was possible to insert a probe into the œsophagus at the stoma, but the defect in the anastomosis was not very convincing. There was a probe hole in the aorta and another in the soft posterior wall of the left main bronchus. The major bronchi were filled with clotted blood and there was a little blood in the œsophagus. Death had undoubtedly occurred as a consequence of bleeding into the respiratory tract. The blood in the œsophagus may have been swallowed or may have entered the œsophagus through a defect in the œsophago-gastrostomy.

Case 8.—A 53-year-old woman who had had dysphagia for 4 months was shown to have a squamous carcinoma of the œsophagus 39 cm. from the upper alveolus. Œsophago-gastrectomy was undertaken in May, 1958, and the œsophago-gastric anastomosis was made at the lower border of the aortic arch. Throughout convalescence she was pyrexial, once to 103 F. (*Fig.* 298). Six years before she had undergone cholecystectomy and convalescence had been complicated by venous thrombosis of the left calf. On the eighth post-operative day after œsophago-gastrectomy the left calf became swollen and tender. Heparin in the dosage outlined in *Case* 2 was administered and the calf rapidly returned to normal. On the eleventh post-operative day she vomited a small quantity of bright red blood and bled a little per vaginam. It was not the time for her normal period and it was thought that the vaginal bleeding and hæmatemesis were

consequences of the administration of heparin: she received protamine sulphate and a transfusion of fresh blood. She died 18 hours later from massive hæmatemesis. At autopsy the œsophago-gastrostomy and the anteromedial aspect of the descending aorta 7 cm. distal to the left subclavian artery were adherent over an area of about 0·5 cm. There was a direct communication between the lumen of the aorta and the alimentary tract through the œsophago-gastrostomy, and the fistula admitted a small probe. There was no intervening abscess cavity. There was no evidence of bleeding elsewhere. Œsophagus and stomach were filled with clot. The pyrexia which this was made, bringing up the stomach medial to the arch. At no time during her convalescence was she regarded as well. She was pyrexial and pleural liquid re-accumulated despite repeated aspiration of the left pleural space. On the eighteenth post-operative day auricular fibrillation developed, the rate but not the rhythm being controlled by digoxin. On the thirty-first post-operative day and on each of the two subsequent post-operative days she had a small hæmatemesis and died from massive bleeding on the thirty-fourth post-operative day. A fistulous communication between aorta and stomach lay 4 cm. distal to the left subclavian artery. The œsophago-gastrostomy

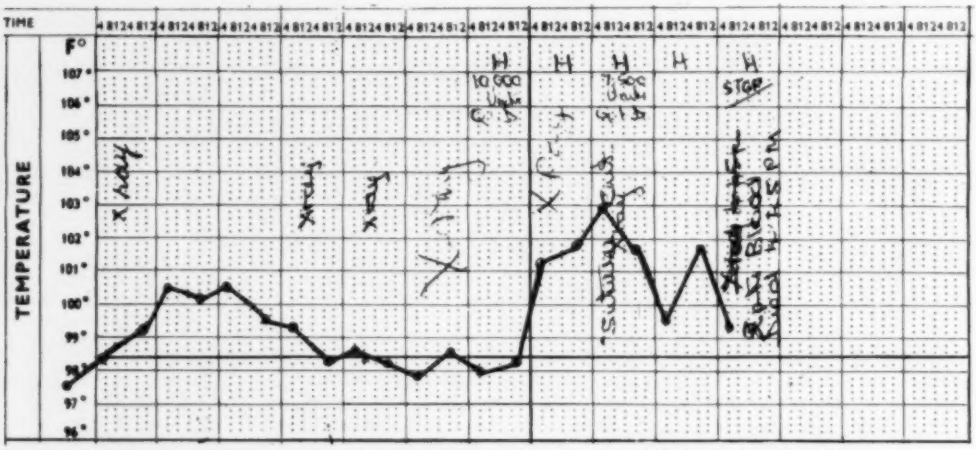

FIG. 298.—Temperature chart from *Case* 8. This patient was given heparin during the last 5 days of life.

patient developed early during her convalescence may have been a reaction to œsophagectomy and the high fever later in convalescence may have been related to the development of venous thrombosis.

Case 9.—A 58-year-old man who had had dysphagia for 5 months was shown to have a squamous carcinoma of the œsophagus 31 cm. from the upper alveolus. Œsophago-gastrectomy was undertaken in September, 1956, and an œsophago-gastric anastomosis made at the lower border of the aortic arch, the anastomosis retracting medial to the arch. Mediastinal glands were invaded by tumour. He was pyrexial until the thirteenth post-operative day, when he developed auricular fibrillation. On the evening of the thirteenth post-operative day he complained of a sensation of tightness in the chest, vomited a small quantity of blood, and half an hour later died from massive hæmatemesis. Death was as much from suffocation from the aspiration of blood as from blood-loss. At autopsy the fistulous communication between aorta and œsophago-gastric stoma was less direct than usual. The hole in the aorta was about 3 cm. distal to the small defect in the œsophago-gastrostomy, and there was between these holes a narrow fibrous-lined track in the mediastinum. The defect in the aorta may have been the site of ligation of an œsophageal artery, because such an artery was ligated at about this level and a silk ligature was found in close proximity to the aortic fistula. There was the usual black slough on the aortic adventitia with a punched-out probe-sized hole in the centre of the slough.

Case 10.—A 48-year-old woman who had had dysphagia for 6 months was shown to have a squamous carcinoma of the œsophagus 33 cm. from the upper alveolus. Œsophago-gastrectomy was undertaken in June, 1950. The aorta was mobilized distal to the left subclavian artery by the division of several intercostals, and a supra-aortic anastomosis was intact and the defect in the stomach was at a distance from the œsophago-gastrostomy—at that end of gastric closure farthest from the œsophago-gastrostomy. There was no intervening abscess cavity, this aspect of the gastric suture line lying directly against the descending aorta.

These 10 cases are summarized in *Table I*.

Case 11.—A woman aged 42 years who had had dysphagia for 2 years and who was shown to have a squamous carcinoma at 28 cm. from the upper alveolus was submitted to œsophago-gastrostomy in March, 1958. She had a very extensive tumour and the œsophago-gastrostomy was made at the lower border of the aortic arch. She became ill from respiratory infection on the sixth post-operative day, and a tracheostomy was made (*Fig.* 299). Two days later it was obvious that she was inhaling ingested liquid. Oral feeding was stopped, but she died on the tenth post-operative day from respiratory infection. At post-mortem examination the œsophago-gastrostomy had partially disrupted and the resulting mediastinal abscess had eroded the soft posterior wall of the left main bronchus, with the formation of a large œsophago-bronchial fistula. This patient is mentioned since the mechanism of development of this fistula must be the same as that in the previous cases.

THE OPERATIVE PROCEDURE AND POST-OPERATIVE MANAGEMENT

The details of the operation of œsophago-gastrectomy as undertaken in the Thoracic Unit in Edinburgh have been described elsewhere (Logan, 1961). Block dissection of the mediastinum includes exposure of the back of the trachea and a considerable length of both stem bronchi, and

stripping the aortic sheath laterally, anteriorly, and medially. When the œsophago-gastrostomy or œsophago-jejunostomy lies at the lower border of the aortic arch or medial to it there is no tissue available in the mediastinum for covering the anastomosis, which lies therefore in direct contact with aorta devoid of sheath. The aorta covered by parietal pleura and penicillin or crystomycin. The pleural drain was usually removed on the first post-operative day. The patients were allowed to drink limited quantities of liquid from the second post-operative day and were usually ambulant by the third or fourth post-operative day. Chest radiographs were made on the first and second post-operative days and thereafter at regular

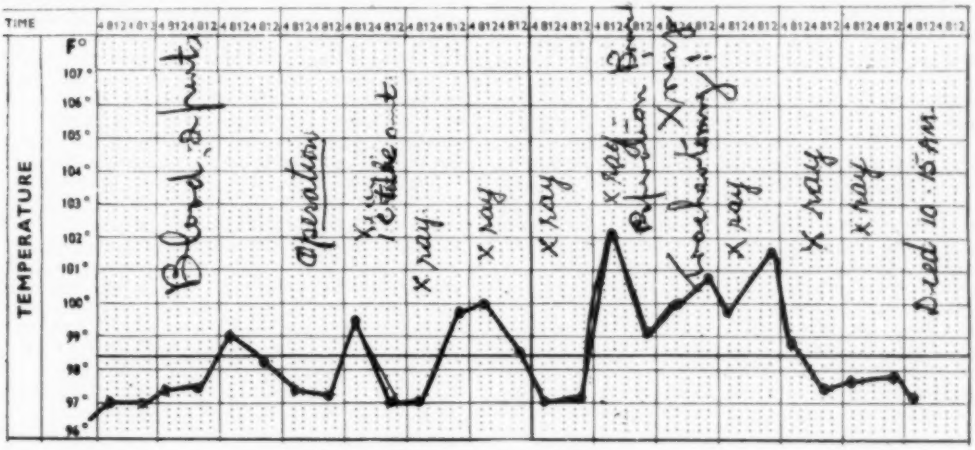

FIG. 299.—The temperature chart from *Case* 11.

Table I.—DETAILS OF 10 CASES

CASE NO.	AGE	SEX	DURATION OF DYSPHAGIA (months)	LEVEL OF PROXIMAL EDGE OF TUMOUR FROM UPPER ALVEOLUS (cm.)	GLANDULAR INVASION BY TUMOUR	YEAR OF OPERATION	REPLACEMENT BY STOMACH (G) OR JEJUNUM (J)	ANASTOMOSIS BELOW THE LEVEL OF THE AORTIC ARCH	SUPRA-AORTIC ANASTOMOSIS	POST-OPERATIVE PYREXIA PERSISTING FOR AN ABNORMALLY LONG PERIOD	WARNING HÆMORRHAGE	POST-OPERATIVE DAY ON WHICH FATAL HÆMORRHAGE OCCURRED	INTERVAL AVAILABLE FOR SURGICAL INTERVENTION FOLLOWING WARNING HÆMORRHAGE	POST-OPERATIVE FIBRILLATION	HEPARIN USED IN POST-OPERATIVE PERIOD	HISTOLOGY OF ŒSOPHAGEAL TUMOUR
1	77	M.	2	41	−	1953	G	+	−	+	+	29	2 d.	+	−	Adenocarcinoma
2	46	M.	2	35	−	1957	G	+	−	+	+	15	5 hr.	−	+	Adenocarcinoma
3	49	M.	3	37	−	1959	G	+	−	+	+	15	8 hr.	−	−	Adenocarcinoma
4	61	M.	3	38	+	1960	G	+	−	+	+	39	3 d.	−	−	Adenocarcinoma
5	68	F.	3	28	+	1959	G	+	−	+	+	14	5 hr.	−	−	Undifferentiated
6	54	M.	3	29	+	1954	J	+	−	+	+	21	30 hr.	−	−	Squamous
7	73	F.	4	24	−	1956	G	+	−	+	+	27	Nil	−	−	Squamous
8	53	F.	4	39	−	1958	G	+	−	+	+	12	18 hr.	−	+	Squamous
9	58	M.	5	31	+	1956	G	+	−	+	+	13	30 min.	+	−	Squamous
10	48	F.	6	33	+	1950	G	−	+	+	+	34	3 d.	+	−	Squamous

sheath is normally very resistant to infection—it is rare for a patient to die from aortic hæmorrhage in relation to chronic pleural infections. Aorta devoid of sheath would appear from the series reported here to be very vulnerable to destruction by infection. The resistance to infection of aorta devoid of pleural covering but not of sheath is not known.

The post-operative management of these patients was in no way different from that routinely undertaken after œsophago-gastrectomy. The resections were undertaken under cover of an antibiotic such as intervals or when there was any specific indication. No tube was passed through the œsophago-gastrostomy and the gastric remnant was not aspirated. In all these cases bowel-sounds had returned by the morning of the second post-operative day. Post-operative fever after the second or third day was managed by the administration of a wide-spectrum antibiotic before adequate investigation of, for example, organisms in the sputum had indicated precise sensitivities on which to base chemotherapy intelligently. Purées and soft solids were being taken

by the sixth post-operative day and normal diet by the tenth day.

PATHOLOGY

The autopsy findings in the 10 patients who died from hæmorrhage and the 1 patient who died with an œsophago-bronchial fistula were very similar. In 2 patients there was an abscess cavity in the mediastinum through which the aorta was found to communicate with a defect in the œsophago-gastric stoma, and in 1 patient there was an abscess cavity in the mediastinum through which the aorta communicated with an œsophago-jejunostomy. In 1 patient aorta and left main bronchus communicated through an abscess cavity, and in this patient there may have been an additional defect in the œsophago-gastrostomy. In 3 patients there was a fibrous track making the fistula between the aortic defect and the defect in the œsophago-gastrostomy, without an abscess cavity. There was a considerable inflammatory reaction in relation to this fistula. In 3 patients the aortic defect communicated directly with some part of the gastric closure or the œsophago-gastrostomy.

Disruption of the anastomosis with the formation of a pyopneumothorax is an occasional dramatic sequel to œsophago-gastrectomy. It is suggested that in these patients dying from hæmorrhage the anastomosis had leaked into the mediastinum much less obtrusively, through a small defect, and an abscess had formed in the mediastinum with subsequent inflammatory erosion of the aorta. The post-mortem appearances in those patients with an abscess cavity support this suggestion.

In those with a fibrous track through the mediastinum the abscess may have been small, may have absorbed or burst back into the alimentary tract, or the inflammatory process initiated by leakage from the anastomosis need not have progressed to the formation of an abscess. In those patients in whom there was direct communication between aorta and stomach an inflammatory process in the mediastinum seems likely, and in these cases particularly it is attractive but probably unjustifiable to postulate that acid gastric secretions may have contributed to aortic digestion.

CLINICAL PICTURE

In only 1 of the 10 patients was convalescence entirely uneventful and in this patient there was no warning hæmorrhage. In the remaining 9 patients there was a fairly consistent clinical picture of convalescence complicated by persisting fever, in 7 without obvious cause and in 2 possibly related to venous thrombosis. Fever without known cause—and a common cause is respiratory infection—is unusual later than the third post-operative day following œsophago-gastrectomy. A warning hæmorrhage preceded the terminal hæmorrhage in 9 patients; 6 of these had not yet been discharged from the surgical ward from which they had been submitted to operation, and in the 3 others the warning hæmorrhage precipitated their re-admission to hospital. The interval between first and terminal hæmorrhages varied from 3 days to 30 min. and in all but one of these patients this time interval was sufficient to make a diagnosis and to intervene surgically with the object of preventing further bleeding.

Two patients bled during a course of heparin. In neither of these could the mediastinal pathology be related to the administration of heparin. It is not possible to determine whether heparin precipitated hæmorrhage. Atrial fibrillation is a common complication of thoracotomy in the elderly, occurring usually in the first 4 post-operative days. Four of the 10 patients in this series had atrial fibrillation, but in only one did this complication develop early—on the second post-operative day. In most patients who have died from disruption of the anastomosis it has been observed that atrial fibrillation precedes clinical recognition of disruption of the anastomosis, usually by 1 or 2 days. It is suggested therefore that the development of atrial fibrillation late in convalescence from œsophago-gastrectomy is a manifestation of mediastinitis and heralds some further disaster.

The patient, therefore, who remains pyrexial without obvious cause and who develops auricular fibrillation later than usual in convalescence—in the second or even the third post-operative week—is very likely to have something wrong with the anastomosis. If that patient bleeds there can be no doubt that the source of hæmorrhage is a large vessel in the mediastinum, probably the aorta, and further that the patient will die from hæmorrhage unless some action is taken. In the 418 resections recorded elsewhere (le Roux, 1961) no hæmorrhage occurred later than 24 hours after resection which did not ultimately result in death. The hæmorrhage is a certain warning of death in the absence of surgical intervention and there is usually the opportunity for initiating such intervention.

SUGGESTED SURGICAL MANAGEMENT

Prevention of this catastrophe is more desirable than the undertaking of heroic surgical procedures with a very limited chance of success. It may therefore be undesirable to denude the aorta of its sheath. Block dissection of the mediastinum is not possible without stripping the aortic sheath, and in the absence of this manœuvre any attempt at radical œsophagectomy may be abandoned. The operative mortality from radical œsophagectomy for localized tumours is in any event as high as for disseminated tumour and the late results—survival 5 years after operation—are little different from those following less radical procedures. In this Unit 70 patients have been submitted to œsophago-gastrectomy for peptic stricture of the œsophagus, and aortic erosion has not occurred as a complication in these patients. In these resections the anastomosis frequently lies in close relation to the lower border of the aortic arch, but the aortic sheath is not stripped during œsophagectomy for a benign lesion. Trouble of any sort with the anastomosis is rare after œsophagectomy for a benign lesion, and this may be related to the fact that the more conservative dissection in the mediastinum leaves the surgeon with material in the mediastinum for covering the anastomosis.

There remains the problem of the patient in whom thoracolaparotomy has been undertaken to restore swallowing and in whom resection of the œsophageal tumour is not possible without wide dissection in the mediastinum, including denuding the aorta of its sheath, opening the right pleural space, perhaps resecting pericardium, and clearing the back of the

trachea and both stem bronchi. The patient in whom such a dissection has been undertaken is then exposed to the risk of aortic erosion in the latter part of his convalescence, and from the figures presented in this series that risk is between 2 and 3 per cent. The number of œsophagectomies undertaken each year in the Thoracic Unit in Edinburgh has been fairly constant over the last 10 years and it will be seen from the dates of the operations in *Table I* that aortic erosion occurs about once every year, during which time 40 or 50 radical œsophago-gastrectomies would be undertaken.

Accepting the denuding of the aorta as one factor predisposing to aortic erosion, leakage from the anastomosis must be another important predisposing factor. The anastomoses in this series have all been made in the same way. Interrupted silk is used, and the anastomosis is made in two layers. The inner layer includes the full thickness of the œsophageal and gastric walls, particular care being taken to include œsophageal mucosa and submucosa, the only substantial structures in the œsophageal wall, and gastric muscularis, because here the mucosa is weak. The sutures are placed at intervals of 2–3 mm., and all the knots are tied on the mucosal aspect. The second, outer, layer is inserted in such a way that the slack gastric wall is drawn up over the first layer, giving the anastomosis the final appearance of an inkwell, or an incipient intussusception. Figures quoted elsewhere show that the incidence of disruption of the anastomosis has not changed significantly over the last 10 years, despite the fact that the surgeons undertaking the resections may have become more adept at making the anastomosis. The basis on which patients are selected for operation has not changed, and many with relative contra-indications to operation undergo operation simply to restore swallowing. If this technique of anastomosis is inadequate, then, in addition to abandoning radical œsophagectomy by not denuding the aorta of its sheath, a different technique of anastomosis may need to be adopted.

There are some patients in whom fever has persisted for many days after resection, and who have developed atrial fibrillation late in convalescence, and who have not developed clinical evidence of trouble from the anastomosis. It is probably unjustified, therefore, to re-explore the mediastinum of a patient simply on the basis of persisting fever and atrial fibrillation. The warning hæmorrhage is indeed a warning since subsequent death from hæmorrhage seems inevitable. From the examination of post-mortem material it is very unlikely that the aortic defect could be controlled other than by resection of a cylinder of aorta and by restoring continuity with a prosthesis. The insertion of a prosthesis into an infected field is an unattractive proposition. If a prosthesis is to be inserted the aorta will need to be occluded proximally and distally, and this procedure will require some by-pass technique, or the operation must be undertaken with the assistance of hypothermia. A convenient by-pass is one in which the tip of the left auricle is cannulated and blood is directed from the left atrium through an occlusive pump into the left femoral artery (Morris, Witt, Cooley, Moyer, and DeBakey, 1957). The rate of by-pass is controlled by the blood-pressure in the arms—if the pressure rises in the arm after cross-clamping the aorta, the rate of pumping is increased, and if the pressure falls the pump is slowed. A filtration pressure of 50–60 mm. Hg is produced in the kidneys and liver by such a by-pass, and this is adequate for a prolonged period of aortic occlusion. The priming volume of such a circuit is small. Aortic occlusion distal to the carotid vessels in the hypothermic patient is probably safe for at least an hour (Cooley and DeBakey, 1956).

The surgical procedure suggested, therefore, for the patient who has given warning of impending death from hæmorrhage after œsophago-gastrectomy is as follows: If a by-pass is available the left hemithorax is opened, the left atrium and left femoral arteries are cannulated, the patient is heparinized, the by-pass started, and the aorta cross-clamped immediately distal to the left subclavian artery and above the diaphragm. The mediastinal dissection is then undertaken; the aortic defect is identified and excised and continuity is restored with a graft, say of dacron. Aortic circulation is then restored. Attention is next directed to the œsophago-gastrostomy. The œsophagus and stomach are likely to be full of blood, making handling of these structures difficult. An œsophageal tube passed by the anæsthetist may help in emptying them. If the defect in the anastomosis is small it can probably be repaired. If the œsophagus and stomach do not hold stitches, or if the defect is large, a new anastomosis will have to be made. At this interval after œsophago-gastrostomy the stomach in the mediastinum is an inelastic, leathery, thick-walled tube. It is quite unlikely once the anastomosis has been excised that the gastric and œsophageal apertures could be approximated to make a new œsophago-gastrostomy without tension. If local repair of the anastomosis is not possible, it will therefore be necessary to restore continuity with jejunum or colon.

If a by-pass is not available, the patient is cooled, the left hemithorax is opened, the aorta cross-clamped as already suggested, and heparin injected into the aorta distal to the caudal clamp.

An attempt has been made to undertake such a procedure in only 1 patient (*Case* 3). There was not the time to cool the patient, and the behaviour of the heart has already been described. The findings on mediastinal dissection in this patient, and on post-mortem examination in the other cases described, make it possible that some success may attend the procedure outlined.

SUMMARY

Ten cases of aortic erosion complicating œsophago-gastrectomy for carcinoma, and terminating in death, are described. The pathology of the lesion is discussed. A clinical picture is drawn, stressing the need for taking cognizance of the warning hæmorrhage. Ways in which this complication may be prevented are discussed, and suggestions for the surgical management of the established lesion are outlined.

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THE SURGICAL ASPECTS OF GASTRITIS AND ITS ROLE IN GASTRIC PATHOLOGY

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GASTRITIS as an entity with surgical implications has gained scant recognition from British surgery, partly because the grosser lesions which have been described by Continental writers are rarely found in our patients, partly because its milder manifestations are successfully treated at home, and partly because its more acute forms usually yield to medical treatment. The absence of radiological peculiarities is another reason for its neglect since the negative report has tended to reassure the clinician.

Nevertheless, cases of gastritis are frequently encountered in surgical practice. The first example is that of a young man with the symptoms of an acute abdomen who is sent for admission as a probable case of acute perforated ulcer but who recovers within forty-eight hours from his attack of acute gastritis and is able to return home without operation. The second relates to a chronic dyspeptic with an acute exacerbation who is discharged after a full gastric investigation with the report that there is no evidence of a peptic ulcer. The third is an older patient suspected of having a gastric carcinoma who is told that he has "only a chronic gastritis".

The purpose of this communication is therefore to draw attention to gastritis as an entity which profoundly affects the health of the individual and has a close relation to peptic ulceration and to carcinoma.

Historical Note.—Knowledge of the gastric mucosa and its secretions dates from 1825 when Beaumont began his experiments and observations upon the gastric fistula of the Canadian St. Martin. His work, published in 1833, contains the first description of the response of the mucosa to irritation. At this time also Robert Carswell (1833–8) at the London Hospital recognized the phenomenon of gastric autolysis and differentiated its post-mortem effects from pathological changes. The drawings of 'follicular' erosions in acute gastritis and of chronic ulcers and chronic erosions in his atlas of *Pathological Anatomy* are therefore the first reliable descriptions of these lesions.

A generation later came the first histological studies of the gastric mucosa in disease, based on sections cut free-hand, compressed between glass, and unstained. We can only wonder that any results were obtainable on such unfixed material. To Hanfield Jones (1852–4) goes the credit of three reports, the most interesting being on the morbid changes in the stomach of an epileptic who died of burns. Wilson Fox (1858), a pupil of Virchow, described acute and chronic gastric catarrh, and in 1880 Samuel Fenwick published his work on gastritis, and related gastric atrophy with pernicious anæmia. Meanwhile the invention of the stomach tube by Leube (1879) and its modification by Ewald enabled the latter to introduce the test-meal (1886) for the study of gastric secretion in health and disease. This led to the detection of anacidity in various conditions and to much speculation about gastritis and its association with gastric ulceration and with carcinoma. The need for unimpeachable histology and for the elimination of autolytic changes prompted Damaschino (1880), Chauffard (1882), and Hayem (1892) to fix the stomach immediately after death with bichromate, but Faber and Bloche (1900) found that 10 per cent formalin was better and this method has survived in pathological work. Faber (1904) elucidated the mucosal changes in all the stages of gastritis and correlated his gastric studies with those of Chauffard (1882) in typhoid fever, of Dieulafoy (1898–1902) in pneumonia and acute appendicitis, and of Hayem and Lion (1897) in acute infections. They recognized the diffuse hæmatogenous form of gastritis and Nyfeldt and Wimtrup (1932) elaborated its progress in fatal cases of diphtheria. Faber's lectures at Guy's Hospital on "Gastritis and its Consequences" were published in 1935 and remain a classic on this subject.

A series of collateral studies based on operation specimens followed Faber's original work, the most notable being those of Konjetzny (1923), Kalima (1924), Orator (1925), Puhi (1926), and Puchert (1931). In this country Sir A. F. Hurst (1929) supported Faber's views on gastritis and Konjetzny's on the relation of gastritis to carcinoma, whilst in America, Bloomfield and Polland (1933) gave support in their exhaustive study on "Gastric Anacidity and its Relations to Disease".

Since the turn of the century, Schindler (1923), Moutier (1935), and Henning (1929), pioneers in gastroscopy, have described the visible lesions of gastritis, and more recently Wood, Doig, Motteram, and Hughes (1949) by the introduction of the flexible gastric biopsy tube have enabled the clinician to take 'blind' samples of mucosa from the body of the stomach for histological study, and thereby to assess the progress of the disease from the antrum towards the fundus.

PATHOLOGY OF GASTRITIS

The term 'gastritis' is used to describe all the non-malignant lesions of the gastric mucosa in which direct invasion by pathogenic organisms can be eliminated.

Two varieties are described: (1) an exogenous or irritation gastritis which is localized primarily in the pyloric antrum and is largely of surgical interest (*Fig.* 300); and (2) an endogenous or toxic gastritis, which, being hæmatogenous, is consequently diffuse and is largely of medical interest (*Fig.* 301). Magnus (1952) has drawn fresh attention to these two forms of acute erosive gastritis. Surgeons are familiar with the irritative variety, but the diffuse type has been largely overlooked, partly because it is often a terminal event and partly because it has not been clearly identified with infections. Yet it is responsible for the 'black vomit' of acute appendicitis and of septic peritonitis and for the late acute hæmatemesis which

may complicate septicæmia, as, for example, in a fulminating acute lobar pneumonia, or toxic states such as develop in uræmia, diabetic coma, and congestive cardiac failure. Faber attributed the anorexia, the more or less profound dyspepsia, and the vomiting, which often accompany acute febrile infections, to a diffuse gastritis. It remains for investigators to demonstrate how much irreparable damage such infections inflict upon the gastric mucosa, for we know that in later life nutritional and blood disorders develop in patients with a diffuse chronic atrophic gastritis.

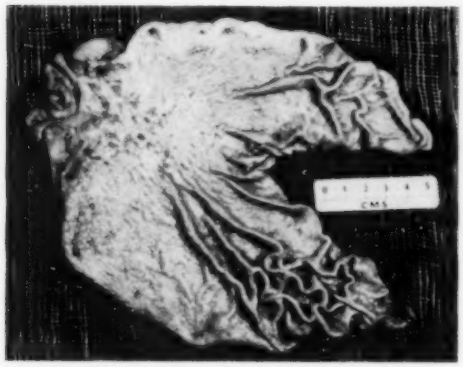

FIG. 300.—Exogenous gastritis: acute erosive antral gastritis.

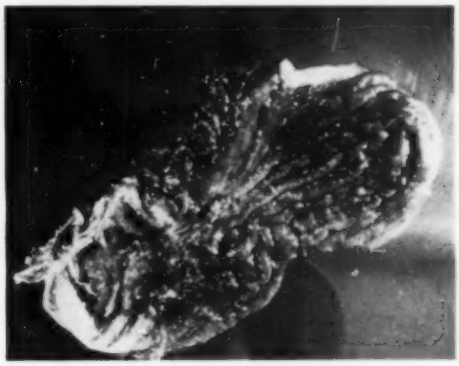

FIG. 301.—Endogenous gastritis: acute diffuse gastritis.

The inflammatory lesion in both varieties is essentially an erosive gastritis which, though generally classified as acute and chronic, cannot be thus artificially divided as it usually occurs in waves, each adding its quota of damage to the gastric mucosa. Once established, gastritis tends to progress step by step to a stage of chronic atrophy.

Acute Gastritis.—In the acute phase the mucosa is red and swollen, with multiple erosions on the ridges and a patchy layer of adherent mucus in the furrows. Microscopically there is an intense cellular infiltration of the deeper layers and a serofibrinous exudate in the superficial layers which is well marked in the gastric tips and wherever the capillaries are dilated and congested. This œdema in the mucosa may well be accentuated by the fact that the muscularis mucosæ acts as a limiting membrane. Magnus (1952) has drawn particular attention to the fluid vacuoles containing polymorphonuclear leucocytes which appear between the cells of the surface epithelium and has suggested that this is the route by which these polymorphs escape to the surface. Another explanation could be that these fluid vacuoles are analogous to the blisters which form in cutaneous inflammation and that in rupturing they not only serve to decompress and so to protect the mucosa, but are in fact the origin of the multiple pinpoint erosions so characteristic of acute gastritis, their greyish-white coloration being due to a deposit of fibrinous material and cellular debris over the lesions.

Larger erosions occur when circumscribed areas of the superficial part of the mucosa and its glands are destroyed by the inflammatory process. The epithelium degenerates and necroses, lymphocytic infiltration and desquamation follow, and the damaged area is shed into the gastric lumen. The deeper part of the mucosa is not breached and healing apparently occurs without scar formation so long as the surface remains in a neutral medium and is protected from acid and pepsin by its normal coating of alkaline mucus. It is also noteworthy that the cells of the surface epithelium undergo alteration: they become more cuboidal, stain darkly, and may form a layer several cells in thickness. Although the initial lesion is in the pyloric antrum, similar changes may also occur in the body mucosa, with surface and glandular erosions, with early disintegration of the chief cells (pepsin), and with some damage to the parietal (oxyntic) cells which, though more resistant, tend also to become disorganized.

Chronic Gastritis.—With each exacerbation further inflammatory changes occur, with more cellular infiltration in the deep layers, more glandular destruction, and a new crop of erosions on the surface (*Fig.* 302). Sometimes a few erosions remain as chronic lesions. Chronic erosions have a warty appearance with pitted centres, their raised edges being due to cellular infiltration. When they heal, a layer of surface cells covers the central depression.

In chronic atrophic gastritis there is still further evidence of glandular destruction in the pyloric antrum and of widespread cellular infiltration. This may result in cyst formation—chronic cystic gastritis (*Fig.* 303)—or in the development of lymphoid follicles (*Fig.* 304)—chronic follicular gastritis—whilst the surface epithelium is replaced by an intestinal type containing many goblet cells and often referred to as 'intestinal metaplasia', which can be shown by special histiochemical stains to secrete mucus differing from that of the normal stomach. The pyloric glands are ultimately destroyed and with them possibly the source of gastrin which is said to be produced within the pyloric antrum (*Fig.* 305). When the body mucosa is affected, glandular destruction is also seen and 'pseudopyloric' glands are formed in an attempt at regeneration, but these do not secrete pepsin. Here, too, areas of intestinal epithelium appear on the surface. Thus the special secretory powers of the body

mucosa are progressively destroyed and a new type of mucus is produced which may lack the full protective qualities of gastric mucus.

In chronic gastritis the mucosa has a flat glistening appearance and following a flare-up of the condition in the first part of the duodenum in acute and chronic antral gastritis and Schade (1958a, b) has confirmed these. To be accurate, we ought therefore to speak of an acute erosive gastroduodenitis and of chronic gastroduodenitis in this disease (*Fig.* 305).

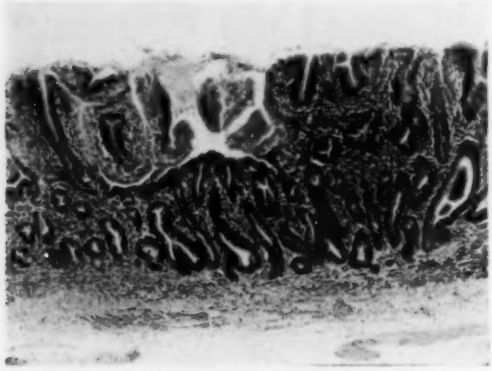

FIG. 302.—Acute on chronic antral gastritis. *Note*: (1) Intense cellular infiltration; (2) Area of superficial necrosis with overlying fibrinous membrane; (3) 'Intestinal metaplasia' of glandular epithelium.

FIG. 303.—Acute on chronic antral gastritis with cystic formation. *Note*: (1) Intense cellular infiltration; (2) Glandular erosion becoming cystic.

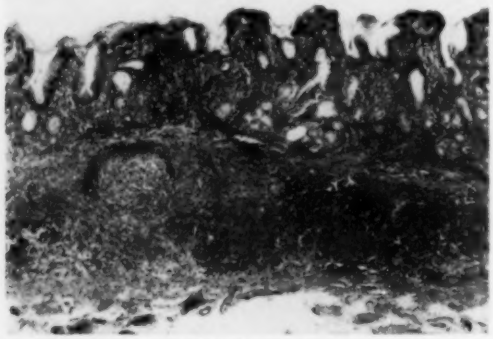

FIG. 304.—Chronic follicular gastritis. *Note*: (1) Atrophic changes in mucosa; (2) Intense lymphocytic infiltration with follicular formation.

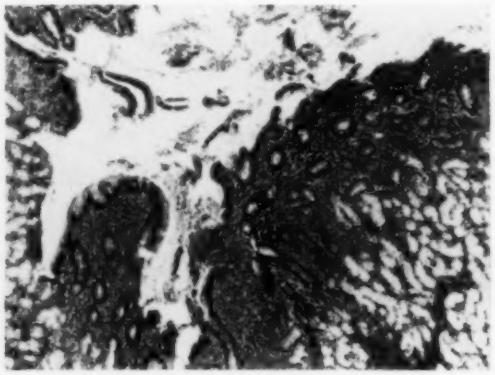

FIG. 305.—Duodenitis. Some atrophy of surface epithelium. Intense cellular infiltration.

there may be evidence of recent erosions in numerous small scattered points of inflammation or of pigmentation from mucosal hæmorrhage. Occasionally one or more chronic erosions may be present (*Fig.* 306).

In the latest stages of the condition the mucosa becomes thin or atrophic and it is replaced by a flat surface layer of mucus cells resting upon a fibrous stroma, largely devoid of glandular elements, extending to and blending with the muscularis mucosæ.

The most remarkable feature of chronic gastritis is its tendency to spread upwards on a broad front from its origin in the pyloric antrum to the body mucosa, so that it too becomes flat and glistening and loses much of its rugosity. The highest point of the gastritic process, therefore, always lies on the lesser curve and it may approach close to the cardia. Konjetzny (1938) described similar mucosal changes

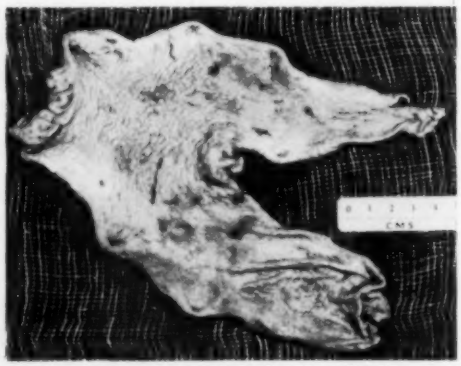

FIG. 306.—Advanced mucosal atrophy. Note chronic erosion on pylorus.

THE STAGES OF GASTRITIS

As long ago as 1891, Fischel classified gastritis as catarrhal, interstitial, and atrophic. Wood and his collaborators used the flexible gastric biopsy tube to study the histology of cases of uncomplicated gastritis (1949). Doig and Motteram (1950) described three stages, each merging into the next, viz.: (1) Acute or superficial gastritis; (2) Chronic atrophic gastritis; (3) Gastric atrophy.

They state that they rarely saw signs of resolution in superficial gastritis, thus confirming Schindler's observation that the inflammatory changes linger on long after symptoms have disappeared.

Later, Weiden and Funder (1952) compared the histology in a series of uncomplicated cases of gastritis with the results of test-meals. They found that as the mucosa of the body of the stomach proceeded from a normal state to that of atrophy, there was a progressive diminution: (1) In the total volume of secretion; and (2) In the concentration of acid and pepsin; and (3) A steady fall in the amount of fasting juice occurred, thus establishing a true biological relationship between the histology and the gastric secretions in this progressive disease.

The importance of chronic atrophic gastritis was emphasized by Schade (1958a, b) when, over two years previously, a patient with symptoms suggesting a gastric carcinoma, but without radiological evidence of it, was reported by him to have gastric cells highly suspicious of a carcinoma. On the strength of some notable successes in the cytological diagnosis of early cancers a partial gastrectomy was done. The patient made a good recovery and has remained well and symptom free. A search throughout the specimen revealed not a carcinoma but a chronic atrophic gastritis with 'intestinal metaplasia', the atypical exfoliated goblet cells having been mistaken for those of a mucoid carcinoma.

It appeared that cytology might be of use in the diagnosis of non-malignant conditions as well as in carcinoma and that there was still much to learn about the surgical aspects of chronic atrophic gastritis.

It was therefore decided to review all the cases of gastritis recently investigated in the hospital in order to re-assess the clinical history of the condition. There were 49 personal cases and 34 others—a total of 83.

THE CLINICAL ASPECTS OF GASTRITIS

Table I shows the ages at which symptoms first appeared and the sex incidence in 83 cases of gastritis.

Table I.—Age and Sex Incidence

Ages	*Males*	*Females*	*Totals*
10–19	3	—	3
20–29	10	1	11
30–39	12	3	15
40–49	16	6	22
50–59	12	7	19
60–69	3	4	7
70–79	4	2	6
	60	23	83

Even from this small group it is clear that gastritis is commoner in males than in females up to 50 years of age, and that thereafter it declines in frequency and affects the sexes equally. The highest incidence is in the fourth decade, but it may occur at any age.

Clinical Types of Gastritis.—Gastritis may be acute, recurrent, or persistent in type, and a recurrence may commence as an acute exacerbation. In the recurrent variety symptoms tend to assume fixed patterns of dyspepsia, but in the more persistent types the symptoms vary both in character and in intensity from case to case.

Table II relates the types of gastritis to the length of the clinical histories.

1. *Acute Gastritis.*—This is usually due to irritation of the gastric mucosa by a large indigestible meal, strange food, or an excess of alcohol, but it may be the result of food poisoning. It occurred in the 1914–18 war immediately after gas attacks, when men swallowed saliva in which irritants were dissolved. Some of these men had hæmatemeses. The onset may therefore be an acute dyspepsia, a gastroduodenal hæmorrhage, or a combination of these two conditions. When the dyspepsia is severe there is epigastric pain, tenderness, and rigidity, with vomiting and often collapse, so that the attack simulates an acute perforation and the patient is admitted to hospital. The pain may be an acute burning sensation or the result of a severe gastric colic. Rigidity is never so complete as in perforation and free gas is not found on radiography. Recovery usually follows in 24–48 hours, when the stomach has lost its irritating contents. If an exploratory operation is done on the mistaken diagnosis of a perforation, intestinal obstruction, or appendicitis, no lesion is found, but the stomach may appear irritable, injected, or œdematous, or quite normal. The treatment is to empty the stomach by tube and aspiration if necessary and to keep it empty, except for such bland fluids as are required to allay thirst. A milk-drip overnight is useful. Rest and warmth are essential. No drugs need be given unless pain is severe. Free hydrochloric acid is not always present in the aspirate, but if it is an insoluble alkali, with milk, cream, or olive oil, and possibly atropin, will help to protect the inflamed mucosa from acid and pepsin.

There were 5 examples of acute gastritis, 3 admitted as acute perforations and 2 as acute exacerbations of ulcer. Dahl of Oslo (1930) reported 4 such exacerbations of gastritis operated upon as threatened perforations and in these acute inflammatory changes were found along with chronic changes, but no ulcers.

Hæmatemesis, melæna, or both, with but more frequently without dyspepsia, may also signalize the onset of a gastritis or of a relapse.

In 1953 the writer (1954) reviewed 225 cases of acute gastroduodenal hæmorrhage and found that 70 were due to acute lesions, only 1 of which required surgical operation. It is of course impossible to say whether the bleeding comes from an acute ulcer, from erosions, or from the multiple minute points of a gastrostaxis without viewing the lesions either by gastroscopy as originally practised by Avery Jones (1947) and Tanner (1949) in selected cases, or by operation in severe cases, since radiology is usually unable to detect acute ulcers. In his series of 63 emergency operations for uncontrolled gastroduodenal hæmorrhage Tibbs (1957) demonstrated that 20 had acute gastric lesions; 11 of these were acute ulcers (5 with associated erosions), 5 were acute erosions, and 2 were diagnosed as gastrostaxis: in addition there were 2 acute duodenal ulcers with

associated multiple gastric erosions. It therefore remains to be shown whether erosions as the primary lesions of acute gastritis cause hæmorrhage more frequently than acute ulcers in the less severe types of bleeding.

gastric hyperfunction was accompanied by hyperæmia, hypersecretion, and hyperacidity. With inflammation and œdema minor stimuli to the mucosa caused pain, its sensitivity being increased by hyperæmia and engorgement, which lowered the threshold.

Table II.—Relationship of Type to Length of History

Length of History	Acute Onset		Recurrent Fixed Pattern Dyspepsias	Constant Atypical Dyspepsias	Totals
	Dyspeptic	Bleeding			
1. Days	3	3	—	—	23
Weeks, months	—	—	9	8	
2. 1–5 years	2*	1*	17	3	23
3. 5–37 years	—	2*	31	4	37
	5	6	57	15	83

* Acute exacerbations.

Table III shows the incidence of hæmorrhage in 83 cases of gastritis.

Thus bleeding alone occurred 17 times and bleeding with dyspepsia 6 times. Control was usually secured by medical measures, but 3 patients required

Table III.—Incidence of Hæmorrhage in Gastritis

Present Attack	*Initial Onset*	*Relapse*	*Totals*	
Bleeding only	3	3	6	17
Previous Attacks				
Bleeding only	4	7	11	
Bleeding and dyspepsia	3	3	6	
	10	13	23	

partial gastrectomies and in each acute erosions were responsible.

Gastrostaxis from a congested mucosa is the most difficult type of hæmorrhage to manage since it may not yield to routine treatment and may persist from the gastric stump after a partial gastrectomy. Daly, Johnston, and Penberthy (1949) recommended the use of phosphate buffer solution and topical thrombin as an adjunct in treatment which they found useful in determining whether surgery was required. They reported successes in arresting hæmorrhage in 90 of 100 cases. It would therefore appear to be particularly suitable for acute erosions. It should not be forgotten that aspirin may sometimes cause hæmatemesis.

Barlow, Bentley, and Walder (1951) demonstrated the rich vascularity of the gastric mucosa and thereby explained how multiple erosions may cause a massive hæmorrhage. Walder (1952; 1957) investigated the arteriovenous anastomoses which they described and showed that each has a central cellular chemoreceptor mechanism like the carotid body, which regulates the supply of CO_2 to the oxyntic cells during activity. There seems therefore no connexion between these anastomoses and disease of the mucosa.

It is important to note that an erosive gastritis need not be painful. Indeed it resembles some acute peptic ulcers which may bleed or perforate without prodromal dyspeptic symptoms.

Ivy (1940) believed that continuous gastric pain was due to congestion, œdema, or inflammation of the mucosa, and that intermittent pain was muscular in origin.

Wolf and Wolff (1943) found that normal gastric mucosa was highly insensitive to stimuli and that

Thus it appears that a mild or superficial gastritis may be painless and that the local digestive processes resulting in an acute ulcer may also be painless, but when the inflammation is greater and spreads to the submucous muscular or peritoneal coats, painful symptoms will always occur.

2. *Chronic Recurrent Atrophic Gastritis* (57 patients).—After the initial attack a recurrence may be long delayed, but relapses tend to recur at shortening intervals and to last a few days, differing thus from the longer relapses of peptic ulcers. They are brought on by indiscretions in food or drink, a severe infection, and sometimes by a period of severe mental or physical strain. There is a marked tendency for the symptoms to assume a fixed pattern which can be related to a phase in the gastric cycle.

a. A filling dyspepsia occurs when symptoms appear during or immediately after a meal. More food aggravates the condition and vomiting follows, with relief.

There were 13 patients in this group.

b. The pyloric mill syndrome is characterized by the onset of symptoms 15 min. or so after food. This corresponds with the beginning of active gastric peristalsis and with the maximal outpouring of gastric juice. There is generally pain, which more food increases, alkali may relieve, and which, if prolonged, is frequently terminated by vomiting.

There were 11 patients in this group.

c. During the emptying phase symptoms resemble either those of a gastric ulcer, or, when more delayed, those of a duodenal ulcer. Referred pains, hunger pains, and night pains also occur. Nineteen patients had a gastric ulcer syndrome and 14 patients had a duodenal ulcer syndrome.

Thus there is no single dyspeptic pattern in recurrent gastritis, but such could hardly be expected in a condition which begins as an acute superficial juxta-pyloric gastroduodenitis and finally encroaches upon the body of the stomach as a chronic atrophic gastritis. Indeed in some patients with long histories, the symptom pattern did change from the acute 'filling' to the chronic 'emptying' dyspepsias, and in several others the symptoms became more severe and more constant.

It is worthy of note that 33 of these 57 patients had ulcer-like symptoms—in fact many had been investigated more than once as possible ulcers, and

even laparotomy failed to demonstrate any ulceration. One patient, a young doctor, with severe recurrent pains, demanded a partial gastrectomy, a procedure which confirmed the presence of an active chronic gastroduodenitis without ulceration and which has afforded lasting relief for over 3 years. Fortunately, symptoms are not always so acute.

In the few cases where hæmorrhage and dyspepsia occurred together, it was noteworthy that the dyspepsia was more frequently of the acute filling and early post-prandial patterns than of the ulcer-like syndromes—a fact which if substantiated in a larger series might be of clinical value in differentiating between the hæmorrhage of recurrent gastritis and that of chronic ulceration.

3. *Late Chronic Atrophic Gastritis.*—The group of 15 patients with atypical symptoms suffer from ill health and are chronically dyspeptic. Here the atrophic process is more advanced and gastric acidity is low or absent. The gastric mucosa is more fibrotic and less elastic and is more firmly fixed to the gastric musculature so that it is incapable of much expansion. These patients complain of a sense of pressure or fullness in the epigastrium, of gas and flatulence, of pain whenever they eat, and they cannot take much food. After solid food the pain may last a long time and they get ease by voluntary vomiting. Others with more atrophy complain of nausea and discomfort after every meal; they lose weight and strength, and are easily tired. Food and alkali do not bring relief. Some become anæmic and have frequently occult blood in the stools. In those with achlorhydria diarrhœa may be an intermittent or a constant feature. They all become stomach conscious, some become introspective, and the odd one may even be psychoneurotic. Most become resigned to their plight and for this reason they may overlook the more rapid decline which heralds the development of a carcinoma.

Physical signs: There may be no physical signs in chronic gastritis, but during an acute exacerbation there may be a diffuse epigastric tenderness in the region of the pyloric antrum. The deep tender spot of a chronic peptic ulcer is absent.

DIAGNOSIS

The diagnosis of gastritis requires a complete gastric investigation. It is based on a careful clinical history and physical examination. The barium meal report will be negative unless the stomach or duodenal bulb is irritable or spastic, and this may confuse the radiological diagnosis. The deformity of a healed ulcer scar may also mislead.

Gastroscopy should confirm the presence of gastritis, but the antrum is not always well seen. Small ulcers or growths not readily localized on the X-ray screen can be eliminated.

We have come to rely upon a cytological diagnosis, and it should exclude malignancy (Schade, 1956). A gastric biopsy by Wood's flexible biopsy tube will provide a fragment of body mucosa for histological examination and this will show how far the gastritis has advanced into the upper part of the stomach.

The fractional test-meal affords a quick check on acidity values but it is not diagnostic in gastritis. Hyperacidity has been found in a few early cases but generally acidity is low or anacidity is already established. The histamine test is used to separate true from false achlorhydria and with true anacidity carcinoma must be excluded. The quantity of mucus withdrawn in the early acute case is small: it is often scanty in gastric atrophy but in recurrent cases it is excessive in amount. Hurst (1929) showed that lavage with weak hydrogen peroxide removed most of this mucus and he reported the re-appearance of free hydrochloric acid in some cases.

Occult blood tests should be done, and if there is anæmia a full blood examination is essential.

Some doubtful cases call for decision by laparotomy, particularly when there is a radiological abnormality or when cytology suggests the possibility of malignancy. Should laparotomy reveal no particular lesion, there may be evidence of gastritis in Schoemaker's 'pyloric blush', in peritoneal thickening or stippling, in perigastric or duodenal adhesions, or in some obliteration of the lesser sac. The pyloric lymph-nodes may be enlarged, soft, and pink. The antrum may be tubular and firmer, and contract more vigorously, than normal. A simple pyloric fibrosis is a rare complication. If no abnormality is seen, a gastrotomy is required to examine the mucosa and a biopsy specimen may be taken. A sterilized sigmoidoscope made air-tight in the opening with a purse-string suture facilitates the inspection of an inflated stomach. The first part of the duodenum may be seen through the pylorus; likewise, the lower end of the œsophagus can be entered and inspected. Through this instrument secretions can be aspirated from dependent parts and long biopsy forceps can reach to any part of the mucous membrane. By this method two early carcinomas have been located and in one case of recurrent hæmatemesis two small bleeding erosions were seen along with a minute ulcer.

TREATMENT

Conservative Measures are indicated in acute episodes and should always be tried first. When symptoms subside it would seem desirable to investigate the stage reached in the underlying gastritis. Medical treatment is still largely symptomatic and until we know from the study of individual cases how the disease progresses and responds to therapy, the problem it presents is unlikely to be solved and the aetiology of recurrences will remain a mystery.

Surgical Measures are required when medical therapy fails, and experience shows that operation is indicated for uncontrolled hæmorrhage, for cases in which early carcinoma is a possibility, and for severe recurrent gastritis with intractable pain.

The subject of chronic follicular gastritis was reviewed by Fitzgerald (1931) when he published 9 cases treated successfully by partial gastrectomy. He recounts that Finsterer (1920, 1921, 1924) was among the first to advocate this operation for cases where the clinical history pointed emphatically to the presence of organic disease but where no ulcer or cancer could be found at operation. His cases and those reported in the literature totalled 90, and partial gastrectomy resulted in 77 cured, 6 improved, and 7 failures. He quotes Wanke's warning (1929) that the failures were due to early fundus gastritis. The writer's view is that unless the whole of the area affected by an active chronic gastritis is removed, including its advancing upper margin, recurrent

gastritis, with pain on eating or soon afterwards, will continue to affect the gastric stump. On the same basis a partial gastrectomy should not be performed on any patient with a fundal or a diffuse gastritis.

It remains for us to examine the association of gastritis with peptic ulceration and with carcinoma, and to determine if possible what its relation is to each of these.

GASTRITIS AND PEPTIC ULCERATION

The fundamental problem in this relationship is whether the gastritis is primary or secondary. It has often been described as secondary, notably in cases where a chronic ulcer is surrounded by congested

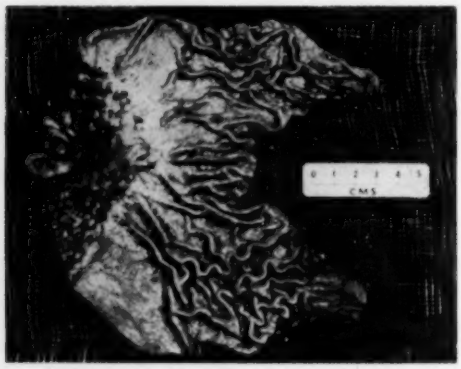

FIG. 307.—Prepyloric gastric ulcer with acute inflammation around it and area of chronic atrophic antral gastritis proximal to it.

mucosa, but closer examination, and in particular histological examination, shows that the chronic gastritic process is widespread and advanced and is not limited to the environs of the ulcer. The evidence, in fact, all points to the gastritis being the primary condition as the following considerations show (*Fig.* 307).

1. Clinical Evidence.—We have seen that in over 50 per cent of the recurrent cases the symptoms simulated those of gastric or duodenal ulcers. Hansen (1932) called them 'pseudo-ulcers' because no ulcers could be demonstrated. In the early years of gastric resection, Konjetzny (1924) and Puhl (1926) found that these cases were a source of diagnostic error. Faber (1927) recognized them as chronic gastritis and he cites (1935) the experience of Nicolaysen (1928), Dahl (1930), Holsti (1931), Roholm (1932), and of Anderson (1935) in support. Similarly, in an even higher percentage of our cases, the tendency to recurrence is as characteristic a feature as is the 'periodicity' of peptic ulceration. Indeed, it seems a reasonable suggestion that these symptoms belong primarily to the gastritic process and that active peptic ulceration only accentuates or modifies the symptomatology.

2. Surgical Experience.—In writing of gastritis and of duodenitis, Sir Hugh Devine (1940) records doing laparotomies on patients with ulcer-like symptoms in whom no ulcers were found, but he states that several of them returned later for operation with chronic ulcers.

This experience is by no means unique and may be contrasted with those examples of recurrent dyspepsia with a radiological deformity, which at operation is found to be due to the healed scar of a former ulcer—commonly duodenal—current symptoms being due solely to the acute exacerbation of a chronic gastritis.

In addition, the writer can recall at least two cases in which a posterior gastro-enterostomy had been done for troublesome 'duodenal' dyspepsia in which at a subsequent laparotomy for recurrent symptoms there was no sign of ulceration in stomach, duodenum, and jejunum and the diagnosis was that of chronic gastroduodenitis.

3. Histological Evidence.—Magnus (1952) has investigated the association of gastritis with gastric and duodenal ulcers. In 93 per cent of 284 gastrectomy specimens for gastric ulceration he found chronic atrophic gastritis localized to the pyloric antrum in 73·5 per cent and diffuse in 26·5 per cent (*Fig.* 307). In the remaining 7 per cent there was an acute erosive gastritis.

In 100 per cent of 256 gastrectomy specimens for duodenal ulceration he found chronic atrophic gastritis milder in degree and localized to the pyloric antrum. Very occasionally there was an acute erosive gastritis.

He observed that the gastritis of gastric ulceration was always severe, and he could not demonstrate any differences in the gastric mucosa of ulcer-gastritis and cancer-gastritis. Again, he stated that this gastritis showed no specific characteristics and resembled in every way the acute and chronic gastritis seen apart from chronic ulceration.

Schade (1958a, b) has confirmed these observations, but he has also shown that an acute or chronic duodenitis accompanies every gastric or duodenal ulcer.

There must therefore be an intimate relationship between the simple gastroduodenitis already described and the gastroduodenitis of peptic ulceration.

4. Pathological Evidence.—

The Formation of a Peptic Ulcer.—It is generally agreed that the chronic ulcer arises from an acute erosion by way of the subacute phase and the small acute round ulcer. Konjetzny described this process and more recently Wolf and Wolff (1943) demonstrated it experimentally by exposing an erosion on the edge of the gastric fistula of their subject 'Tom', where it lacked adequate cover from protective mucus, to the continuous action of the gastric juice for 4 days. A typical peptic ulcer formed in its place. In addition, the gastric mucosa became injected and there was a significant increase in the quantity of acid secreted. Thus a vicious circle was established. By protecting the ulcer from further digestive action it healed in 3 days, the inflammation subsided, and the secretion diminished in amount. They concluded that in the presence of a denuded surface on the mucosa a chronic ulcer would result from the interplay of protective and destructive forces.

The Combination of Acute and Chronic Lesions.—Schade (1958a, b) has reported that gastrectomy specimens show every combination of acute and chronic lesions in peptic ulceration, and an extensive personal experience confirms this (*Figs.* 308–310).

In duodenal ulcers, erosions and even small acute ulcers are frequently found on the mucosa of the pyloric antrum and occasionally of the body.

In active chronic gastric ulcer scarring and multiple acute lesions occur in the stomach and duodenum.

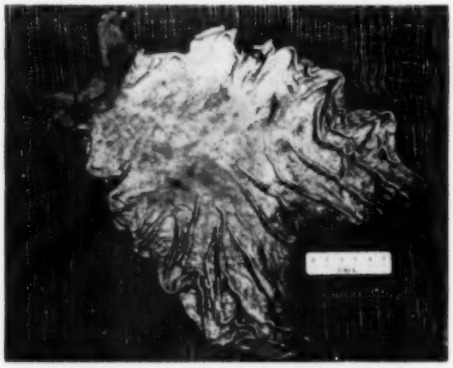

FIG. 308.—Inflamed chronic duodenal ulcer with extensive chronic atrophic antral gastritis.

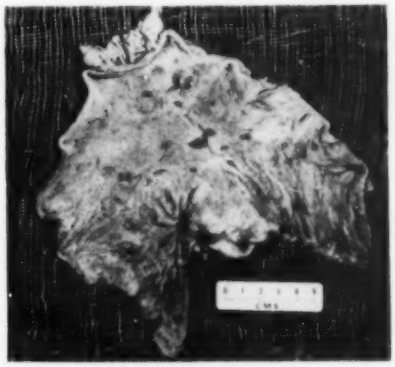

FIG. 309.—Two high lesser curve gastric ulcers with acute erosive antral gastritis.

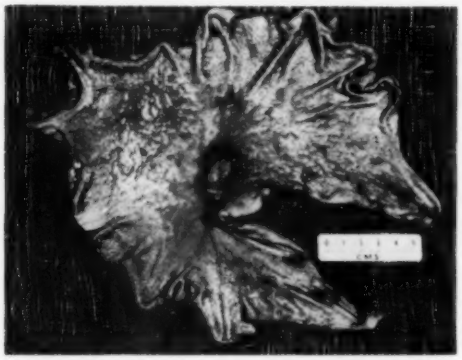

FIG. 310.—Large lesser curve gastric ulcer with marked glistening atrophic antral gastritis.

FIG. 311.—Surface carcinoma arising in area of atrophic gastritis. *Note*: (1) Overlying adherent cellular membrane; (2) No penetration deep to muscularis mucosæ.

In chronic gastric ulcer, chronic gastritis and chronic duodenitis can always be demonstrated.

In chronic duodenal ulcer the pyloric antral mucosa shows the presence of intestinal epithelium in 70 per cent of cases, and in chronic gastric ulcer the percentage is even greater.

In chronic ulceration the gastroduodenitis is also chronic, and the severity of the lesions appears to be directly proportional to the length of history and the time which has elapsed from the last exacerbation.

In other words, whilst gastroduodenitis is an entity in itself subject to acute exacerbations which cause a progressive degradation of mucosal structure, these recurrent attacks may be complicated by the appearance of acute erosions or acute ulcerations, and in time by the development of chronic ulceration in the duodenum, at the pylorus, or higher up in the stomach. When recurrences occur in the presence of an established chronic ulceration, the mucosa may show acute erosions and even acute ulcers and the chronic ulcer itself becomes acutely inflamed. In the intervals these lesions all tend to heal, leaving tell-tale scars as evidence of former attacks.

These facts seem to indicate that gastroduodenal ulceration is an incident in an inflammation primarily involving the mucosa of the first part of the duodenum and the pyloric antrum and that therefore acute and chronic peptic ulcerations are secondary to gastroduodenitis. If this is true, the explanation why some patients develop duodenal ulcers, others gastric ulcers, and some both, must lie in the nature of this inflammatory process.

Gastritis and the Situation of Chronic Gastric Ulcers.—A few gastric ulcers are prepyloric and have much in common with duodenal ulcers, but the great majority occur on or near the lesser curvature and on the proximal edge of the pyloric antrum. Reference has already been made to the upward expansion of chronic antral gastritis on the body mucosa and to the fact that the highest point lies on the lesser curve, this being the shortest wall of the stomach. When a high lesser-curve ulcer is found, even adjacent to the cardia, it lies just within the upper limit of this expansion. Similarly, when more than one chronic ulcer is present in the middle of the stomach they also lie near but within the advancing gastritic edge. In

contrast, the healed scars of former ulcers occupy a more distal position.

This implies that a chronic ulcer always develops on an area of active chronic gastritis, that this region is more vulnerable than the more completely atrophic mucosa lower in the stomach, and that peptic ulceration never develops on a normal mucosa.

Coincident Chronic Gastric and Duodenal Ulcers.—In 1926 Sir David Wilkie drew attention to the presence of coincident gastric and duodenal ulcers. In a series of 300 personal gastrectomies for peptic ulceration he found:—

221 with one or more chronic duodenal ulcers	74 per cent
37 with chronic gastric ulcers	12 per cent
42 with coincident gastric and duodenal ulcers	14 per cent

Thus, in a total of 263 chronic duodenal ulcers, there were 42 with associated gastric ulcers—an incidence of 16 per cent, whilst in a total of 79 chronic gastric ulcers there were 42 with an associated chronic duodenal ulcer—an incidence of 53 per cent. He concluded that in peptic ulceration the first part of the duodenum was the most probable site, and then the lesser curvature of the stomach.

Assuming, as we have sought to prove, that each ulcer is associated with a gastroduodenitis which is initially more superficial in duodenal ulceration and more advanced in gastric ulceration, the higher incidence of duodenal ulceration suggests that it occurs earlier in the disease process along with a lower incidence of concomitant gastric ulceration, whereas gastric ulceration, which is normally of later development, is associated with a higher incidence of duodenal ulceration.

This is in accord with Doll's assessment (Doll, Jones, and Buckatzsac, 1951), for he states that in England duodenal ulcer is found 4 or more times more often than gastric ulcer under the age of 35 years, but that after 45 years it is only 1–2 times as common.

It seems reasonable to conclude that the inflammatory and secretory factors which favour the development of duodenal ulcers operate first, and that later in the disease process they become modified to those associated with gastric ulcers. In the transition period both types of ulceration may be found.

This conception fits with the milder and more localized gastroduodenitis found in duodenal ulceration and with the more severe type found in gastric ulceration. It also offers an explanation for those perplexing differences which characterize duodenal and gastric ulcers. Finally the varying incidence of gastric and of duodenal ulcers at different times and in different countries may well be related to variations in the prevailing conditions of life, social and dietary habits, and other factors which directly or indirectly affect the health and vitality of the gastric mucosa.

CHRONIC GASTRITIS AND CARCINOMA

Konjetzny (1913) was the first to teach that chronic atrophic gastritis was a precancerous condition and to produce histological evidence of the transition from gastritis to carcinoma (*Fig.* 311). Orator (1925) and Puchert (1931) agreed that the gastritis in carcinoma was more advanced than in peptic ulceration and was diffuse. In his Schorstein lecture, Hurst (1929) supported Konjetzny's view from his own extensive clinical experience and stated that 70 per cent of carcinomas developed on the basis of a chronic gastritis which was diffuse and associated with achlorhydria. Bloomfield and Polland (1933), in discussing anacidity and carcinoma, concluded that:—

1. The type of gastritis associated with cancer of the stomach was identical with that found in people with unexplained anacidity.

2. It was entirely gratuitous to assume that a small cancer, perhaps submucous, could produce a gastritis.

3. No specific report existed to prove that an alleged progressive deterioration in gastric acidity occurred in carcinoma.

Therefore "they must agree with Matthieu's (1889) long neglected view that cancer is prone to occur in stomachs already the seat of chronic change".

Magnus (1952) confirmed the presence of chronic atrophic gastritis in each of 95 gastrectomy specimens for carcinoma. In 83 primary growths it was antral in 72 and diffuse in 11, and in 12 ulcer–cancers it was also antral.

In 146 gastric cancers the writer (1958) found that the majority were situated in those parts of the stomach most commonly affected by chronic gastritis, there being 58·7 per cent on the pyloric region and lesser curve, 29·3 per cent on the body and 12 per cent near the cardia.

If we associate carcinoma with chronic gastritis it is fair to compare their clinical histories.

In 30 per cent of 146 carcinomas half of the patients had histories of 1–5 years and half of them histories of many more years. 14·6 per cent were 'ulcer–cancers' but all had the recurrent type of dyspepsia found in chronic gastritis and in peptic ulceration. But in 70 per cent the history was 1 year or less and averaged 5–6 months. There are two possible explanations for such short histories:—

The first is that patients are forgetful. A lady of 72 years admitted with hæmatemesis gave a history of indigestion lasting 3 months. Her cytology revealed malignant cells and radiology showed a small antral polypus. Operation showed not only an early carcinoma in situ high up on the body but in addition the healed scars of both a duodenal and a gastric ulcer. The polyp was simple. She now remembers consulting a physician over three years ago for indigestion. She had a widespread chronic gastritis.

The second reason for short histories may well be that the chronic gastritis is diffuse and endogenous in origin. This type is often completely latent. Achlorhydria is usually present and symptoms are few until the growth begins to affect the patient. Further experience of these cases aided by cytology should enable the pathologist to determine how often endogenous gastritis occurs in cases of gastric cancer.

A three years' experience in Newcastle of early carcinomas suspected clinically and revealed only by cytology numbered 25 cases and in each of them the development of the growth on the basis of chronic atrophic gastritis has been confirmed beyond doubt (*Fig.* 311).

THE GASTRIC PROCESS IN GASTRIC PATHOLOGY

Since duodenal ulcer, gastric ulcer, and carcinoma are each associated with gastroduodenitis at some

stage in its evolution, it should be possible to examine the conditions within the stomach when these lesions commonly develop.

In this connexion the recent paper of Heinkel and Henning (1957) is particularly significant. Using Wood's gastric biopsy technique and test-meals they studied in 946 patients the relationship between the histology of the body mucosa and gastric acidity in each type of peptic ulcer and in carcinoma, including 89 patients with no history of gastric disease as controls. Their report falls into three sections:—

1. The first section is concerned with specific lesions. They confirmed that in duodenal ulcerations acidity values were usually raised or normal. They showed that in carcinoma achlorhydria occurred in 50 per cent and true anacidity in 43 per cent of all cases. They found that in gastric ulcerations acidity values fell into an intermediate range. These secretory levels mean that duodenal ulcers normally occur early in the disease and before the body mucosa is damaged, that gastric ulcers may appear early but more commonly at a later stage, and that 50 per cent of carcinomas develop on an atrophic mucosa.

2. The second section deals with the frequency of different levels of acidity in the various stages of the gastritic process.

They found that as the mucosa of the body of the stomach proceeds from a normal state to that of atrophy there was:—

a. A steady fall in hyperacidities from 48·5 per cent to 5 per cent.

b. A steady fall in normal acidities from 32 per cent to 5·5 per cent.

c. A steady rise in anacidities from 12 to 81 per cent and in true anacidities from 5 to 61 per cent.

3. The third section is complementary to the second in that it shows the incidence of normal body mucosa, superficial gastritis, and atrophic gastritis in each level of acidity. They found that:—

a. The percentages with normal mucosa fell as acidity declined.

b. The percentages with atrophic gastritis rose as anacidity increased.

c. The percentages with superficial gastritis were fairly evenly distributed between the different levels of acidity.

CONCLUSION

These findings are in accord with those of Weiden and Funder (1952) in uncomplicated gastritis.

1. They demonstrate the same biological relationship between histological structure and secretory values and prove that the gastritic process is fundamentally the same whether it is simple or complicated by the presence of peptic ulceration or carcinoma.

2. They confirm that this gastritis is essentially a progressive disease which in its course alters both the structure of the gastric mucosa and the chemical composition of its secretion.

3. Finally, they show that the conditions favourable to duodenal ulceration are to be found early in the disease, that those for gastric ulceration extend to a later stage of the process and that those associated with carcinoma mainly occur in its later stages. It is true that some carcinomas develop in the presence of acid and pepsin on a chronic antral gastritis, e.g., ulcer-cancers and localized carcinomatous ulcers, but the diffuse and infiltrating types and the papilliferous growths are associated with anacidity and an atrophic gastritis.

There appears therefore to be abundant evidence of a clinical, pathological, and chemical nature for the belief that surgical gastritis is a clinical entity and that it plays a primary role in a wide field of gastric pathology. Against such a slowly changing background it is possible to see an explanation for the occurrence of those various combinations of simple and of malignant lesions which are so perplexing in gastric pathology. For these reasons gastritis or gastroduodenitis deserves closer study and its treatment calls for renewed consideration.

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CUTANEOUS AMŒBIASIS

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What is Cutaneous Amœbiasis?—Do amœbæ invade the skin? Nasse (1891) described invasion of the skin and subjacent tissues by the *Entamœba histolytica*, and Craig (1944) pointed out that these invasions occurred at sites at which amœbic abscesses of the liver had been drained, and in the perianal skin. These cases are uncommon, and Kreutzer (1951) was able to collect the records of a little less than 50 cases, in most of which there was a paucity of detail. Kreutzer confirmed the findings of Craig as to the sites at which the lesions occurred.

The Nature of the Lesion.—The lesion is a spreading necrosis of the skin and its subjacent tissues giving a circular orifice opening into a cavity lined by necrotic tissue. The margins of the orifice are raised and indurated and the cavity extends widely and relentlessly with increasing toxæmia. Death is the usual outcome. The appearances suggest that there has been a spreading necrosis of the skin and the tissues subjacent to it from a point to which a deeper-seated abscess had reached. The appearances are quite different from the channels made by pus discharging on to the surface from a deep-seated abscess. It would appear as if there had been a spreading invasion of the skin and subjacent tissues because of the amœbæ finding this a more suitable site for infiltration of the tissues than in the deeper parts of the track of the abscess.

Demonstration of Amœbæ in the Necrotic Tissues of Cutaneous Amœbiasis.—Craig (1944) described the invasion of the skin and subjacent tissues by the *E. histolytica*. The demonstration of amœbæ in sections of tissue removed at post-mortem might not show the amœbæ on account of the disintegration of their protoplasm after death. Even tissues removed by biopsy need careful staining to show the amœbæ. In the cases reported on in this paper, biopsy specimens were stained by the technique described by Silva (1960), of the Department of Pathology, University of Ceylon. The tissues bordering the cavity showed numerous amœbæ.

What are the Chances of Spontaneous Rupture of an Amœbic Abscess on to the Surface of the Body?—An amœbic abscess of the liver will reach the surface of the liver as it enlarges, and it will then rupture through the capsule of the liver. The pus will escape into the peritoneal cavity where it will either flow freely into the cavity or collect in a pool which is walled off by adhesions from the rest of the peritoneal cavity. An amœbic abscess can bulge the muscles of the loin or it can bulge the muscles of the anterior abdominal wall, and it will get larger and larger without eroding a passage outwards through the parietal peritoneum. Amœbic abscesses of the liver commonly bulge into the intercostal spaces overlying the right lobe of the liver, but erosion of the abscess at this site through the muscular fibres of the diaphragm does not occur. Here again the parietal peritoneum walls off the pus very effectively. Amœbic abscesses presenting under the central tendon of the right cupola of the diaphragm not uncommonly erode into the right pleural cavity and even through the base of the right lung into a bronchus, but even here pus does not erode through the parietal pleura to reach the surface of the body over the right lower chest. The spontaneous rupture of a liver abscess on to the surface is an exceptional event, and cutaneous amœbiasis is therefore not a hazard of amœbic abscess provided the abscess is not drained externally.

Does Surgical Drainage of an Amœbic Abscess cause Cutaneous Amœbiasis?—Amœbic abscesses of the liver have been drained and healing has been uneventful. Jordan (1955) and Schapiro (1956) advocate the open drainage of liver abscesses. Their series of cases were small. Heimburger (1925) reported a spreading necrosis of the skin and subjacent tissues following drainage of a liver abscess. How does drainage favour amœbic infiltration of the skin and subjacent tissues? The pus from an amœbic abscess might be teeming with amœbæ or it might contain no amœbæ at all. It might have no amœbæ on the first day of drainage but it may subsequently have many amœbæ. Pus does not drain

FIG. 312.—*Case* 1 on admission to the General Hospital, Colombo. The large crater in the right loin and the sodden tissues below and medial to it are well shown.

through a tube after a few hours, but there is an escape of pus between the tube and the tissues surrounding the tube. The invasion of the skin and subjacent tissues suggests that the amœbæ find these tissues favourable for invasion. Drainage of pus by incision and closure of the wound would be a lesser hazard for cutaneous amœbiasis, and aspiration would be the least hazardous of all methods of removal of pus. At most surgical centres aspiration of pus is preferred to drainage which is reserved for the cases in which the pus is too thick to flow. It is not generally known, however, that if the pus does not flow when it is aspirated, it will not flow when it is drained, the reason being that the pus cannot be aspirated because the necrotic areas in the liver are solid, with the consistency of cheese. Open drainage cannot drain more from such a lesion after the liquid core has been aspirated.

Cutaneous Amœbiasis affecting the Perianal Tissues.—These cases manifest a similar necrosis; they develop a crater with indurated edges opening into a cavity in the ischiorectal fossa; toxæmia is marked and death is usual. Where did the lesion come from in such cases? These cases have a colitis with amœbæ in the stools, and it is likely that the amœbæ invade the ischiorectal fossa in the same way that pyogenic organisms invade it. When the abscess works its way to the perianal skin and discharges spontaneously, the stage is set for amœbic infiltration of the skin and adjacent fat.

CASE REPORTS

Case 1. **Amœbic Invasion of the Skin and Subjacent Tissues following Drainage of an Amœbic Abscess Bulging the Right Loin.**—K. P. N., male, Sinhalese, 28 years old, was admitted on May 27, 1960, to the Kandy Hospital for a large abscess bulging the right loin.

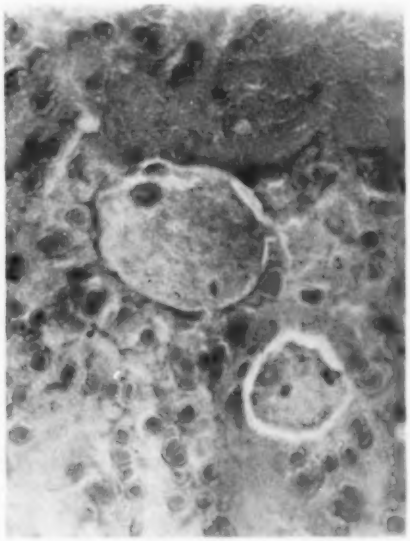

FIG. 313.—Biopsy of scraping from the margin of the crater in the right loin. Two amœbæ with ingested red blood-cells are seen in the granulation tissue. (× 640.)

The abscess was diagnosed as a large perinephric abscess and it was drained on May 30 through a 1-in. incision. Two pints of chocolate-coloured pus were drained from the abscess, and a drainage-tube was put into the cavity. A culture of the pus gave a growth of *Bacillus alkaligenes* and sensitivity tests showed that it was very sensitive to penicillin and sensitive to other commonly used antibiotics. Pus continued to drain from the abscess cavity even though treatment with massive doses of penicillin was given. The patient's general condition deteriorated and he was transferred to the General Hospital, Colombo, on June 30.

At this time he was emaciated to the bone; there was a crater 2 in. in diameter with an indolent raised edge opening on the right loin and leading into a cavity which extended down to the peritoneum. The bare 12th rib projected into the cavity, and the lateral edges of the latissimus dorsi and erector spinæ muscles projected into the lumen on the medial side. The skin medial to the crater over the erector spinæ muscle was sodden and necrotic (*Fig.* 312).

Despite his emaciation and extreme weakness, the patient was able to give a clear account of himself. Four months ago he had pain in the right loin and this was

followed a few days later by high fever which continued as an intermittent temperature. It was evident that the chocolate-coloured pus from the abscess was pus from an amœbic abscess of the liver, and that there was now extensive invasion of the skin and tissues at the site at which the abscess had been drained. The patient was put on intensive courses of anti-amœbic therapy (emetine gr. 2/3 daily; emetine bismuth iodine tablets gr. 1 t.d.s. and chloroquin tablets 150 mg. t.d.s.; terramycin 1 capsule 6-hourly). The temperature ranged between 98·4° and 102° F and he died on July 5. A biopsy was taken from the exposed lateral edge of the latissimus dorsi muscle, and a scraping from the edge of the crater on July 4.

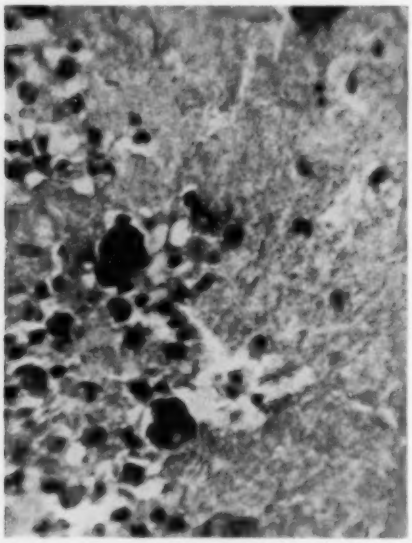

FIG. 314.—Biopsy of portion of latissimus dorsi muscle exposed in the crater. The Silva technique shows the amœbæ darkly stained. The section shows the granulation in the interstitial tissues of the muscle. (× 640.)

REPORT ON BIOPSY SPECIMENS.—

1. From scraping of margin of crater. Large numbers of amœbæ with ingested red blood-cells, and with nuclei characteristic of the *E. histolytica* (*Fig.* 313).

2. From exposed edge of latissimus dorsi muscle. Muscle-fibres diffusely infiltrated with mononuclear cells; zones of necrosis on surface of muscle; large numbers of *E. histolytica* in the necrotic zone (*Fig.* 314).

POST-MORTEM EXAMINATION.—The crater in the right loin was 6 in. in diameter. It had a rim of firm granulations covered by hyperplastic epithelium. The crater opened into a cavity lined by gelatinous sloughs. The 12th rib projected into the cavity devoid of even its periosteum. The edge of the latissimus dorsi was seen exposed. The ascending colon and the lower pole of the kidney were visible in the floor of the cavity. Extending from the edge of the crater from the lower ribs to the iliac crest, and from the midline posteriorly to the mid-axillary line in front, was an area of softening, the overlying skin being necrotic. The muscles were œdematous and covered with gelatinous sloughs.

The abdomen was opened through a midline incision. The peritoneal cavity was clean and dry. There were filmy adhesions of omentum and small intestines below the right lobe of the liver (*Fig.* 315). There were 6 abscesses in the liver, 1 reaching the inferior surface of the left lobe. One of the abscesses reached the right lateral and 2 the superior surfaces of the right lobe. The diaphragm was adherent to one of the abscesses on the superior surface of the liver. The abscesses had a purulent core and a wide zone of solid necrosis. The right lung was adherent to the diaphragm and the right lower lobe was consolidated where it was adherent to the diaphragm. The abscess in the right lobe near the loin had discharged along a track which led to the cavity in the right loin with the large crater on the surface. There were no lesions in the colon and rectum. There was a small area of necrosis in the surface of the brain in the right frontal lobe abutting on the superior longitudinal sinus.

FIG. 315.—The viscera of the trunk removed at post-mortem from *Case* 1. The cut face of the liver shows five solid necrotic masses in the liver. The lowest necrotic mass has extended through the abdominal wall to open on to the surface in the right loin. The necrosis has also extended upwards along the muscle of the diaphragm and the overlying abdominal and chest wall to invade the inferior edge of the lower lobe of the right lung.

HISTOLOGICAL EXAMINATIONS.—These were made from the following sites:—

1. The muscles in the softened tissues around the loin cavity showed infiltration with round cells but no amœbæ.

2. Sections of the walls of the liver abscesses showed an inner zone of granulation tissue and an outer zone of fibroblasts. Within these zones there were pus cells and cellular debris. A few amœbæ were seen in the pus. Beyond the zone of fibroblasts was a zone in which fibrous tissue had infiltrated between the liver cells, disrupting its architecture. There was proliferation of bile-duct epithelium at this place.

3. The lower lobe of the right lung showed filling of the alveoli with plasma cells and lymphocytes. There were areas of necrosis in the lung. Amœbæ were seen both in the necrotic zones and in the alveoli.

4. The area of softening in the brain showed an area of necrosis surrounded by plasma cells and lymphocytes. Fragmented bodies resembling amœbæ were seen.

These histological examinations show that, in cutaneous amœbiasis, necrosis spreads to the surface from a deep-seated lesion. Muscle-fibres resist this necrosis, but the interstitial tissues around the muscle-fibres are affected.

Case 2. **Cutaneous Amœbiasis of the Perianal Region.**—V. H. S. A., 34 years of age, male, Sinhalese, was admitted on July 14, 1960, for an abscess discharging on the surface in the left perianal region, extending from the anal margin to the ischiorectal area, and incessant diarrhœa. The patient was emaciated and dehydrated

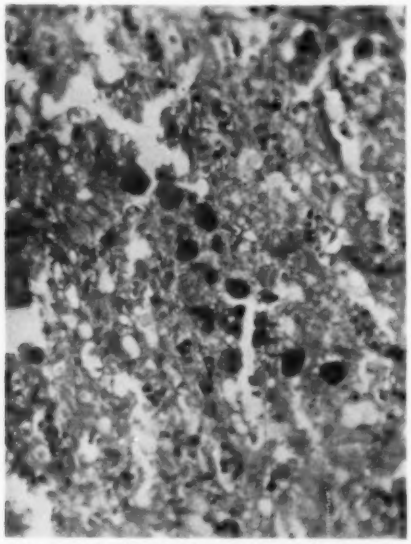

FIG. 316.—Section of the lower edge of the lower lobe of the right lung. The section shows a pneumonic consolidation with numerous amœbæ in the alveoli. Silva technique. (× 320.)

and was treated with mist. bismuth sedative, intravenous fluids, and penicillin and streptomycin. The general condition improved after 24–48 hours although the diarrhœa was uncontrollable. He was treated with sulphaguanidine 1 g. t.d.s. and pulv. cretæ aromat. 10 g. t.d.s. with only a mild response. There was a wide sloughing ulcer in the left ischiorectal fossa. On July 17 treatment was altered to aureomycin 250 mg. 6-hourly together with starch and opium enemata, and chalk and opium mixtures orally, with no improvement.

July 21: The diarrhœa was still incessant. Stools report: No amœbæ, ova, or cysts. No salmonella or shigella organisms.

The resident physician considered that the man was suffering from tuberculosis and put the patient on streptomycin 1 g. daily, oral streptomycin, and INH. These drugs allayed the diarrhœa to some extent.

Aug. 4: A smear and scrapings from the ulcer both showed amœbæ. Treatment: Emetine gr. $\frac{1}{3}$ and strychnine gr. $\frac{1}{60}$ for 10 days and enterovioform 0·5 g. t.d.s. Streptomycin and INH were continued till Aug. 27. Locally: Paste of emetine bismuth iodide, acidi boric, and glycerin.

Aug. 24: Diarrhœa ceased. All drugs were omitted. The perianal ulcer was clean but there was much discharge from it. The patient was gaining weight at a rapid rate.

Sept. 13: Developed diarrhœa again. Treated with emetine bismuth iodide gr. 1 t.d.s. for 10 days and terramycin 250 mg. 6-hourly, 40 capsules.

Sept. 21: Stools showed hookworm ova.

Sept. 28: Abscess almost healed.

Sept. 30: Patient discharged from hospital.

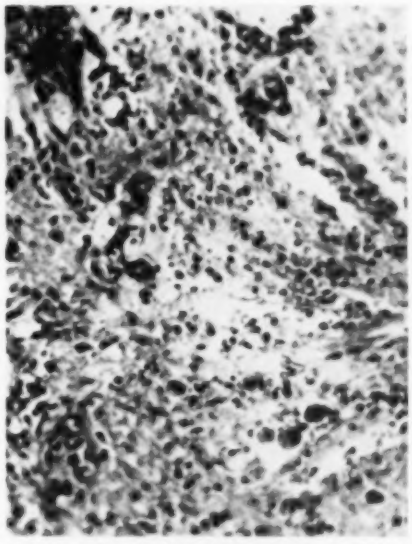

FIG. 317.—A section from the edge of the crater in *Case* 2. At the left-hand bottom corner is the epidermis of the perianal skin. Granulations extend right up to the epidermis and the amœbæ (darkly stained by Silva technique) are seen in the granulations. (× 240.)

Acknowledgements.—Mr. P. D. S. Amarasekera, Photographer to the University of Ceylon, took the photographs illustrating this article. The technique of Mr. E. M. Silva (1960), Technician, Department of Pathology, University of Ceylon, gives the clearest demonstration of amœbæ in tissues of any method that we have used, and this method has been used in these investigations. Dr. S. Y. D. C. Wickremasinghe, Research Assistant to the Professor of Surgery, University of Ceylon, looked after both the cases mentioned in this report and helped in the investigations.

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EXTERNAL CHOLEDOCHODUODENOSTOMY

AN EVALUATION OF 125 CASES

By W. M. CAPPER, Bristol

External choledochoduodenostomy is an operation which, although it has been practised for well over 70 years, has had varying degrees of popularity, and has never been widely accepted. The uncertainty has probably arisen from the fact that, although small groups of cases have been spasmodically reported from various centres, no large group has been analysed to determine the long-term effects, both on the common duct itself, and secondarily on the liver. Moreover, certain cases have been quoted from time to time where the common bile-duct after choledochoduodenostomy has become occluded by food debris such as tomato skin, leading to intermittent jaundice and fever. Most surgeons have therefore been very cautious about the operation. Obviously a follow-up on this operation must first of all determine whether the stoma remains patent; previous reports seem to have neglected this aspect. The following survey of 125 cases, operated on between 1947 and 1960, has been carried out over the last 3 years in order to determine the above and other factors, and generally to evaluate the operation. The average interval between operation and review was 5 years 3 months, the longest 10 years and 3 months, and the shortest 12 months.

HISTORY

Riedel was the first to report the operation in a brief paper produced in July, 1888 (1888a, b). Plenk (Plenk and Hartl, 1949; Hartl and Raindl, 1951) of Lenz, in 1949, reported on 95 cases he had treated privately, with 1 post-operative death. Seventy-one of these cases had a frankly purulent cholangitis secondary to stones. He also described 293 hospital cases and states that no case of post-operative ascending cholangitis had been encountered in any of these. He emphasized that purulent cholangitis would be cured if the stoma was large enough, as the most important thing was drainage. Becker Bernhard (1950) insisted that purulent cholangitis was not an ascending infection from the duodenum but a descending, and that all cholangitis is rapidly cured when the bile can escape freely. He regarded the operation simply as the right way of draining an abscess.

Finsterer wrote several papers on the subject from 1932 onwards (1932, 1942a, b, 1952), and in 1952 reported enthusiastically on 233 cases where the operation was carried out largely for suppurative cholangitis, or when white bile was present. Many of these cases were done at the time of famine œdema, and yet his mortality was 12 per cent. This was less than his mortality for drainage of the common duct, which was 21 per cent, and for transduodenal choledochotomy, which was 27 per cent. He noted that, with a follow-up of from 3 to 25 years, the late results were good and no ascending infection was observed. Three were reported not cured and in 2 cases the stoma had closed. He found that the late results were better than after biliary drainage or transduodenal exploration of the ampulla. He emphasized that the operation gave a lower mortality and better long-term results, and that it was essential for the stoma to be 2–3 cm. in length.

Sanders (1946), reporting on 22 cases, was impressed with the results because the operation was simple, physiological in principle, and gave a wide margin of safety and an easy convalescence. Hosford (1957) reported 19 cases in 1957, and the next year Iszak and Steiger (1958), in analysing 66 cases, noted that choledochoduodenostomy gave fewer complications than sphincterotomy. Schwartz, Benshimol, and Hurwitz (1959) used this method and found it satisfactory in treating 5 cases of stenosis of the lower part of the common duct. They regard hypertrophy and stenosis of the sphincter as part of the general thickening of the common duct following chronic cholangitis. Czyzewski (1961) reports from Poland that he has found the operation so satisfactory that he now does a choledochoduodenostomy whenever he incises the common duct. Indeed, he reports the operation as giving "infallible, immediate" success. Starr (1953) reported that he had used it with satisfaction 75 times in benign, and 20 times in malignant conditions. He describes it as an admirable operation. Hafner (1959), in reporting 41 cases, found no unfavourable effects, and recommended it (as also does Czyzewski), especially if it is not certain that all stones have been removed from the common duct. Mallet-Guy and Descotes (1955), reporting on 100 cases of choledochoduodenostomy done between 1938–53. found excellent and good results in 95, improvement in 1 case, and recurrences, due to stricture occurring in a small stoma, in 4 cases. They state that in most of these cases the operation risk was poor, owing to depressed liver function, recurrent stones, or other illnesses.

OPERATIVE DETAILS

The operation does not present any special difficulty. It is essential that the stoma should be at least 2·5 cm. long, and more if possible. The operation, therefore, should never be done unless the common duct is sufficiently large for this purpose, and in practice a transverse diameter of 10 mm. or more is essential. The gall-bladder should be removed in all cases. The *lowest* part of the supraduodenal portion of the common duct is exposed. Trouble may be experienced by a leash of vessels running transversely or obliquely across it, and these should be undersewn at the edges with fine catgut. The duodenum is then retracted downwards to expose the duct at the point where it passes deep to the pancreas (*Fig.* 318). A vertical choledochotomy incision of about 3 cm. is made as low as possible and right down to the margin of the pancreas, and the duct explored in the usual manner. Priestley, Taylor, and Rogers (1955) suggest that if a small elliptical-shaped portion is excised from

the common duct, the stoma is more likely to remain patent. We have not used this procedure so far. A transverse incision with the diathermy is then made in the duodenum to lie over the ductal incision and the

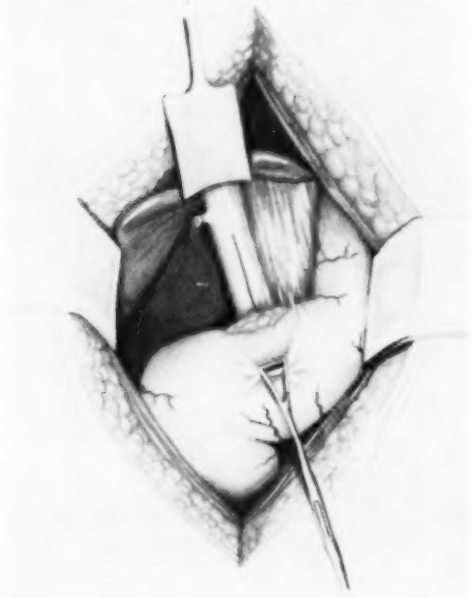

FIG. 318.—One-inch vertical incision as low as possible in supra-duodenal portion of common bile-duct.

anastomosis made with interrupted fine catgut (*Fig.* 319). When finished, the stoma should admit the tips of two fingers, and not just the tip of the little finger (Sanders, 1946).

Sometimes if the first part of the duodenum is unduly tethered, it may be necessary to mobilize the second part and to open the common duct on the right side so that a lateral choledochoduodenostomy may be done (*Fig.* 320).

A few non-absorbable sutures fix the highest part of the duodenum to the fascia over the common duct. A drain is inserted to the subhepatic space. Fung (1961) states that, from his experience with the operation for liver fluke when the duct is dilated, a transverse cut 1·5–2 cm. long is preferable, as it ensures a wide stoma at the lowest possible level. The post-operative course is usually easy. An antibiotic may be given for 48 hours until a good biliary flow is established. The drain can be removed on the fourth, and the patient is usually out on the tenth, post-operative day. In this series there has been no post-operative leak, and the convalescence has been easier and less tedious than when a T-tube drain has been inserted.

In 2 of the cases a piece of rubber tubing was left in the stoma with a view to maintaining its patency. In the first it appears to have been passed without incident. In the second a further operation had to be done 13 months later, when the tube was found impacted 5 ft. down the jejunum.

The operation has been carried out in many cases of advanced age: 7 of the patients were over 80 years; 29 were between 70 and 80 years; and 34 were between 60 and 70 years of age.

FIG. 319.—Commencement of anastomosis with interrupted catgut stitches of fine catgut.

OPERATIVE MORTALITY

The operative (hospital) mortality in this series was 5 cases:—

A. C., female, aged 46 years. Emergency operation for acute hæmorrhagic pancreatitis—died 6 weeks later. Autopsy showed massive necrosis of pancreas.

E. R., female, aged 86 years. Ten weeks' jaundice, carcinoma of pancreas; did not recover consciousness after operation. Died 3 days later.

S. W., male, aged 53 years. Carcinoma of pancreas with gangrene of gall-bladder—liver metastases, deeply jaundiced. Died 2 weeks after operation.

K. V., male, aged 78 years. Gall-stones, gangrenous gall-bladder, general peritonitis. Died 2 weeks post-operatively.

In each of these cases, the primary condition was extremely serious in itself, and it is doubtful whether recovery would have ensued whatever had been done.

The remaining case, a man aged 69 years, developed urinary obstruction with infection 10 days after choledochoduodenostomy for multiple stones in the common duct. Renal failure ensued, and he died at the seventh week. His operation wound had healed and he had no biliary symptoms.

METHOD OF FOLLOW-UP

Altogether 125 cases were considered for follow-up. They had all been operated on by surgeons in the Bristol clinical area. Thirty-one of these cases had died since operation, and their history up to the

time of death and the cause of death were ascertained. The others, 94 in all, were interviewed, either at home or at hospital, and questioned, especially about any biliary symptoms such as jaundice, pain, fever, rigors; then a barium meal was carried out in order to assess stomal patency; liver function tests were

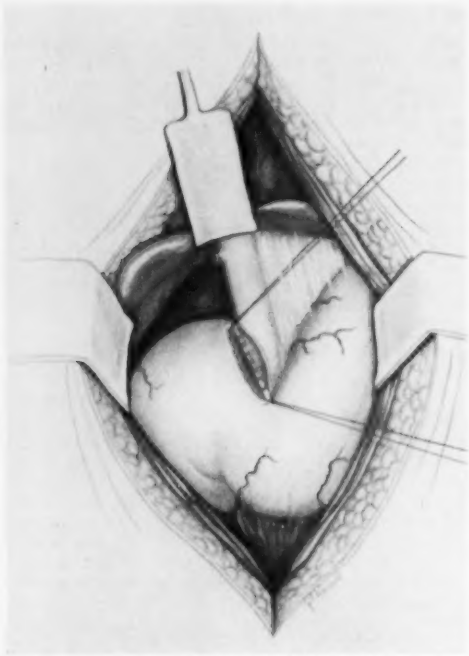

FIG. 320.—Procedure for lateral choledochoduodenostomy.

done, and, in certain cases, a leucocyte count. When these findings were to hand, the patients were again interviewed, and their symptoms finally assessed. A number of cases could not be persuaded to have a barium meal—some of these were over 80 years of age or lived too far away. On the whole, co-operation was good, especially considering the age-group.

DEATHS

Twenty-six cases had died after leaving hospital. Information in all cases was obtained from the practitioner concerned or from autopsy reports.

Table I.—CAUSES OF DEATH

Cardiac	9
Road accident	1
Hypertension	2
Alimentary carcinoma	1
Carcinoma of bladder	1
Chronic bronchitis	1
Cerebral hæmorrhage	2
Cerebral tumour	1
Carcinoma of pancreas	8
Hospital deaths	5
	31

Eight cases in which the operation had been done for carcinoma of pancreas survived an average of 11 months—the longest for 1 year 8 months, the shortest for 2 months. Each case became clear of jaundice following operation, and none had rigors or evidence of ascending infection. Accurate evidence as to whether jaundice returned in the terminal stages was unobtainable, and the attempt was abandoned.

Eighteen cases died of disease unconnected with the biliary tract (*Table I*). The average survival in this group was 4 years 1 month. In none of these cases was there a return of jaundice or any recorded biliary pain or fever before death.

PATENCY OF STOMA

As far as possible, all cases had a barium meal to assess stoma patency; many had been having a barium examination every year in routine follow-up in order to assess, amongst other things, whether the common duct shrinks in size following a decompressing operation. Seventy-two cases, or 76 per cent of the total, agreed to barium meal examination.

Of the 72 cases examined, all but 5 showed complete patency, with barium flowing into and out of the biliary tree. Three showed a filling defect which suggested that there was a residual stone. No case was investigated earlier than 1 year after operation. The average interval after operation was 5 years 9 months. *It may be presumed therefore that in the majority of cases the stoma remains patent.*

The 5 cases mentioned above must be considered separately and in some detail, as they show what happens if the stoma does not remain patent.

M. M., female, aged 35 years. Operation, November, 1954. Cholecystectomy. Gall-bladder contained small stones. The common bile-duct was found to contain stones but was blue in colour and thin. In spite of the narrow diameter, a choledochoduodenostomy was done. In November, 1957, she developed jaundice and fever, and in January, 1958, at laparotomy, we found a stoma 3 mm. in diameter and a narrow common bile-duct which contained tomato skin and debris. These were removed, the duct drained with a fine T-tube, and the duodenum closed.

We consider that in the presence of a narrow common bile-duct the anastomosis should not be done. Starr (1953) says that the real indication is where the duct is dilated from obstruction.

B. D., female, aged 63 years. Operation, March, 1952. Stone in gall-bladder and also in a dilated common bile-duct. There was stricture of sphincter of Oddi. A choledochoduodenostomy was done with thread. Size of stoma not recorded. One year later the stoma was patent to gas only and not to barium. She has had recurrent pain and jaundice since 1955, with rigors at times. Two recent barium meals have evoked the remark by the radiologist, "The degree of patency of the choledochoduodenostomy is doubtful. A ? small amount of gas is present in the biliary tree. No barium will enter."

This patient has the worst possible outcome of the operation. Food debris passes in through a small stoma and does not pass out again, causing blockage. Possibly a small stoma was made, or it may be that biliary deposits had developed around the thread stitches, causing stenosis. Unfortunately she will not agree to further surgery.

E. P., female, aged 56 years. Operation, May, 1952. Cholecystectomy and choledocholithotomy in presence of obstructive jaundice: at forty-seventh day there was a persistent biliary leak and a choledochoduodenostomy was carried out. The biliary leak slowly lessened. She says she

now has shivers from time to time, with back pain. The stoma is, however, not patent to 2 barium meals, and must be presumed to be partially or completely closed.

A. E., male, aged 71 years. Operation, October, 1959. Deep jaundice, white bile, common duct full of stones, with a large stone impacted in the lower end which it was impossible to remove completely. Choledochoduodenostomy. In August, 1960, jaundice recurred with rigors. On re-operation the stoma was found to be closed, the common duct was filled with stones and debris, and there was stenosis of the ampulla. A sphincterotomy was done and the duct emptied. He was well in January, 1961. The operation was done by a surgeon without previous experience of choledochoduodenostomy, and it is almost certain that the stoma was too small. In any case, with a stone impacted in the ampulla, a sphincterotomy would seem to be the right course.

A. R., male, aged 54 years. Operation, February, 1954. Choledochoduodenostomy for jaundice and stone in the common duct. Seven years later no barium enters the hepatic tree, and the stoma must be presumed to be closed. He is perfectly well, apart from symptoms from a hiatus hernia. The note on the operation is the essence of brevity, and no mention is made of the size of the stoma; this man will not agree to a second examination, and the stoma must therefore be regarded as closed. In 3 other cases barium has only been made to pass the stoma on the second radiographic examination.

These 5 cases have been excluded from further consideration and analysis in this paper. In 4 of the 5 cases the operation was a failure, and in 2 cases has definitely made the patients worse. The question arises why tomato skins and food debris should pass into the common duct and stay there in some cases and not in others? Plenk and Hartl (1949), Bernhard (1950), Finsterer (1952), and Mallet-Guy and Descotes (1955) consider that this is due to a small stoma, and this certainly accords with our experience. Starr (1953) thinks that the function of the stoma is directly related to the technique of the anastomosis, and uses interrupted 0000 silk sutures. We consider absorbable sutures to be safer. Stammers (1960) has recorded a case where tomato skin got through a previously undisturbed sphincter up into the common duct. On inspection at operation the sphincter showed no abnormality and bougies passed through it without obstruction. Horton (1961) had a case where tomato skin got into the gall-bladder and to the common duct via a fistula formed by the penetration of a duodenal ulcer into the gall-bladder. Numerous cases have been reported of tomato skin in the gall-bladder following cholecystduodenostomy. If the choledochoduodenostomy stoma is wide, barium can be seen to enter and pass out quite rapidly under an X-ray screen. It is suggestive of trouble if the stoma only admits gas, or if barium passes in and remains there for 24 hours.

RECURRENCE OF JAUNDICE

Each patient was specifically questioned and examined for recurrence of jaundice, and in 66 cases a serum-bilirubin estimation was done. With the exception of the 3 cases of inadequate stoma mentioned above, and the cases dying of carcinoma of pancreas, *no recurrence of jaundice was encountered.* Altogether, and excepting the malignancies, 72 had jaundice either at the time of operation or in the few weeks previous to operation, and in no case did this recur.

PAIN

The group were also questioned about pain which might have been of biliary origin. Excluding again the cases of malignancy, inadequate stoma, and 5 cases mentioned below, no patient actually complained of pain. On closer questioning, however, 9 cases admitted to a transitory pain in the epigastrium and region of the right costal margin. The pain lasts from 10 sec. in the shortest up to 20 min. in the longest case. It was quite sharp, and 2 patients said it was "like the old pain". In 2 cases it was eased by bending over, in another by holding the breath, and in another by coughing. In these 9 cases the operation had been done in 6 for stones, in 2 for relapsing pancreatitis (pain lasting 3 sec. and 20 min.), and in 1 for trauma (pain lasting 2 min.). In no case was the pain regarded as serious, and indeed its presence was only mentioned when questions were specifically asked on the matter. Presumably this must be due to some degree of temporary blockage of the common duct; this is also suggested by the fact that one patient said she had pain after eating fruit.

Five cases call for special mention:—

R. W., male, aged 60 years, started attacks of pain suggestive of biliary colic in 1951, which have remained ever since. In that year a cholecystectomy was performed. The pain persisted. In 1952 a choledochoduodenostomy was done—there were two small stones in a blue dilated common duct. The pain was unaffected by this operation and remained till 1960 when he had a sudden hæmatemesis. At operation a duodenal ulcer was found in association with a widely patent choledochoduodenostomy stoma. Following a Polya gastrectomy his pain has ceased.

M. D., male, aged 62 years, who in 1954 had a cholecystectomy and in 1958 had a choledochoduodenostomy for stenosis of the last centimetre of the common duct. He still has occasional colic, which he finds is relieved by taking sodium taurocholate.

D. W., female, aged 65 years, has had an attack of pain lasting 3 hours, after eating oranges. She had the operation for recurrent stones one year ago.

J. B., male, aged 57 years, had a cholecystectomy in 1957 and a choledochoduodenostomy in 1959: persistently complains of a pain under the right costal margin, which has not been affected by either operation.

J. C., female, aged 53 years, had dragging pains beneath the right costal margin and was diagnosed as a post-cholecystectomy syndrome; her pain has been unaffected by choledochoduodenostomy.

LIVER FUNCTION

Liver function tests were carried out on 65 cases when the stoma was known to be patent. The tests consisted of conjugated and total bilirubin, alkaline phosphatase, total serum-proteins, electrophoretic pattern, thymol turbidity, and flocculation. One case showed raised levels for the alpha-2 and the gamma-globulins. This was a case of relapsing pancreatitis which had jaundice at the time of operation. In the other 64 cases examined no case of depression of liver function was found. Indeed, 1 case improved so much following operation that details will be given.

E. P., female, aged 68 years. History of gradually increasing jaundice with depressed liver function. Albumin 1·7 g. per cent. November, 1952, at operation, had deep jaundice, ascites, white bile in a common bile-duct blocked by one small stone. Nine years later she is active and well, with normal liver function tests and a patent stoma.

Six cases in this series had white bile; none of them shows abnormal liver function tests at the present time. Musgrove, Grindley, and Karlson (1952) found in their cases of choledochoduodenostomy that anastomotic stricture and not intestinal biliary reflux was the main source of liver damage. They also found that slightly, though perhaps not significantly, better results were obtained with choledochoduodenostomy than with choledochojejunostomy. Rodgers and Hancock (1961) have been investigating cases of choledochoduodenostomy by liver biopsy. I am grateful to be allowed to report on some of their work. In 5 cases where the stoma permits free entry and exit of barium, biopsy has not shown any significant changes. One case which had well-marked features of biliary cirrhosis on biopsy at operation in 1954, now has normal biopsy findings. Havard (1960) has shown chronic cholangiohepatitis as a result of stones, with chronic infection and obstruction of the common duct. Whether such biliary cirrhosis is progressive in the presence of an adequate choledochoduodenostomy must be determined; he suggests that infection and not obstruction is the more acceptable factor. Sherlock (1958) states that disturbed hepatic structure and function following biliary obstruction is potentially reversible. Provided that choledochoduodenostomy does not give rise to bouts of ascending infection it should cause improvement rather than deterioration in liver function.

ASCENDING INFECTION

All cases were questioned about fever or rigors, and the response was negative except in the 3 cases of stomal obstruction described above. Five cases, however, remarked that they tended to shiver more following operation than before. The point was not investigated further. It may have occurred at the onset of gastro-intestinal infection. A leucocyte count was done in 10 cases where there had been purulent cholangitis at operation, and this was again negative. We could find no evidence that ascending infection occurs in the presence of an adequate stoma.

INDICATIONS FOR SURGERY

(*Table II*)

The whole series was analysed according to the indications for surgery. Of these by far the commonest was choledocholithiasis.

1. Stones in the Common Bile-duct (79 cases).—Under this head were included all cases reported as having mud, sand, or debris in the common duct.

In nearly all the cases the duct was dilated and in most the wall was white in colour and thickened. In several cases a purulent cholangitis—and in 2 cases liver abscesses—were present. Altogether 79 cases were operated on primarily for stones in the common bile-duct, and, for these, 22 had had previous biliary duct surgery and therefore had residual or recurrent stones. The average follow-up after choledochoduodenostomy in this group was 5½ years. Presumably this should have given adequate time for some recurrent stones to have shown themselves. Apart from the pain detailed above, none of the cases had any symptoms or signs suggesting recurrent stones.

Hughes (1955) found that out of 36 cases of recurrent or residual stones, 32 had caused symptoms within 3 years of operation. Eight of the 36 patients had to have a third operation for stones, and another was admitted moribund with a stone jammed in the ampulla. Some of the 79 cases of our series must have had residual stones—these may have passed through the stoma or still be lying dormant in the retroduodenal portion of the duct. Some of these may be the cases complaining of transient pain, but, if so, it is unlikely it would be relieved by taking a deep breath or bending over.

Table II.—INDICATIONS FOR SURGERY

			Cases
Primary stones			57
Residual or recurrent stones			22
Carcinoma of pancreas			10
Pancreatitis	Acute with stones		1
	Relapsing	without stones	4
		with stones	5
	Chronic	without stones	4
		with stones	5
Dilated duct (history of stones)			8
Trauma			5
Liver fluke			1
Choledochal cyst			1
Post-cholecystectomy syndrome			1
Post-gastrectomy biliary obstruction			1
		Total	125

Some observers consider that stones re-form in a common duct which has stenosis of the lower end. Some degree of stenosis is not an uncommon finding in choledocholithotomy. The cause of this is not immediately apparent. Trommold and Seabrook (1950) considered that the thickened white tough fibrous wall of the common duct, which is such a common finding in choledocholithiasis, also causes fibrosis and stenosis of the sphincter. Cattell, Colcock, and Pollack (1957) consider that partial obstruction due to stenosis of the sphincter may be the cause of the further development of stones. Wangensteen and his co-workers (Imamoglu, Perry, and Wangensteen, 1957), however, suggested that the anatomic narrowing of the terminal common bile-duct was more likely to be the precursor rather than the end-result of gall-stones. They produced fibrosis and narrowing of the distal common bile-duct in animals, and showed that the resultant bile stasis strongly favoured the formation of stones. Clinically they showed that both at operation and autopsy, taking a 3-mm. probe as the significant size, stenosis was rare in the absence of stones and common when they were present. They concluded that though infection is not a necessary component of gall-stone formation, partial obstruction of biliary outflow is. Consideration of the findings in this series would support this conclusion. Choledochoduodenostomy should relieve such obstruction to outflow.

2. Pancreatitis (19 cases).—In the follow-up of such cases, Priestley and others (1955) pertinently remark on the difficulty of evaluating results owing to many variables, such as biliary tract disease, previous operations, degree of pancreatic disease, etc. We have also experienced this difficulty in evaluation and have therefore concentrated on the cessation of attacks and the relief of pain.

Acute Pancreatitis.—There is one case in the series, and death occurred at the sixth week, as described above.

Relapsing pancreatitis was the indication for surgery in 9 cases. The diagnostic criterion in each case was severe pain associated with a serum-amylase above 1000. Of these, 5 had concomitant stones in the common duct. As might be expected, they had no further trouble. Four, however, had a normal biliary tree and merit special attention.

W. W., male, aged 49 years. 1949: Laparotomy: acute pancreatitis. 1951: Cholecystectomy and removal of stones from common bile-duct, but relapsing pancreatitis continued—serum-amylase 1600 in attacks. 1957: Choledochoduodenostomy—no stones found: he has remained asymptomatic for the last 4 years.

D. H., male, aged 45 years. Severe recurrent pancreatitis over 3 years: serum-amylase between 2700 and 1600. 1954: Choledochoduodenostomy—no stones in biliary tree: gall-bladder appeared normal, not removed. No further attacks. Put on 5 st. in weight: remarkably well: asymptomatic for last 7 years.

C. J., male, aged 65 years. 1953: Operation. Jaundice due to recurrent pancreatitis: fat necrosis present, subacute pancreatitis, normal biliary tree—choledochoduodenostomy; gall-bladder not removed—asymptomatic for last 7 years.

M. N., female, aged 49 years. 1958: Relapsing pancreatitis over 2 years: serum-amylase 4000 in attacks; jaundice in present attack. At operation normal biliary tract: gall-bladder removed (normal histology). Choledochoduodenostomy. No further attacks for last 3 years.

It must be emphasized, of course, that a follow-up of 3 years is inadequate in such a disease as relapsing pancreatitis. Priestley and others (1955) regard 5 years as the necessary interval.

Out of the welter of theory and research concerning the aetiology of acute and relapsing pancreatitis, it is difficult, if not impossible, to discern any one determining mechanism. Sinclair (1959), however, states that acute pancreatitis occurs almost exclusively in people whose pancreatic duct and bile-ducts have a common opening into the duodenum. This arrangement was found in 20 out of 21 in the fatal cases of his series. Jackson France and his associates (France, Perkins, and Edwards, 1959) conclude that between 60 and 70 per cent of the population have a common channel. Doubilet (1958) gives a figure of up to 80 per cent.

From the point of view of the operating surgeon, a frequent feature on opening the abdomen in acute pancreatitis is a greenish exudate around the pancreas in spite of the fact that the common bile-duct is intact. The free peritoneal fluid frequently contains bile, and Sinclair (1959) states that at autopsy the bile staining of the pancreas and retroperitoneal tissues is a common finding.

Doubilet (1958) states that under maximal stimulation the pressure on the biliary tree and in the pancreas rises to 300–400 mm. of water, but under normal conditions is usually around 150 mm. The mean intraluminal pressure of the duodenum is approximately 100 mm., with a maximum of 130 mm. of water. Connell (1961) considers that the resting differential between the common duct and the duodenum is about 50 mm. of water. The resting tone of the sphincter is generally considered to be more than 50 mm. Thus a patent choledochoduodenostomy stoma should permit constant dribbling of bile into the duodenum.

When Elliott and his co-workers (Elliott, 1955; Elliott, Williams, and Stewart, 1959) infused the pancreatic duct with bile at 400 mm. water, they found that the pancreas resists infiltration with normal bile and no pancreatitis resulted. If, however, bile was incubated with pancreatic juice for 12–48 hours before infusion, then large amounts enter the pancreas and fatal pancreatitis resulted uniformly. Thus bile activates trypsinogen to trypsin, which destroys the protective action of the biliary mucoproteins.

If this really is the important factor, the practical problem arises as to where the biliary and pancreatic juices are incubated to form the fatal brew? Popper (1942) showed the presence of pancreatic enzymes in the gall-bladder of 10 per cent of people without pancreatitis, but it was present in 16 out of 18 cases who had pancreatitis. Would such incubation take place if these juices were escaping freely into the duodenum? Could such extensive exudation into the pancreas occur with a pressure differential between the duodenum and the common bile-duct of at least 50 mm. water and probably very much more? These factors of course are the physiological rationale for the operation of sphincterotomy. When the sphincter is sectioned, the residual resistance to flow is about 100 mm. water (Doubilet, 1958). Newman and Northup (1957) state that in order for a sphincterotomy to be complete, not only the fibre of the papilla should be divided, but also the more powerful choledochal muscle. This might make sphincterotomy a hazardous procedure in routine use.

Jones, Smith, and Gregory (1958) have shown that following the usual type of sphincterotomy, morphine still produces constriction of the lower end of the common bile-duct as a result of contraction of the duodenal wall. They considered this the reason why sphincterotomy for recurrent pancreatitis was frequently ineffective. Eiseman, Brown, Virabutr, and Gottesfeld (1959) have found that sphincter incompetence following sphincterotomy does not last for longer than 6 weeks. This is an important observation, and they consider that this may in part account for the ineffectiveness of sphincterotomy "noted by so many" in preventing recurrences of clinical pancreatitis. Eiseman and his colleagues, by a study of 27 dogs and 5 humans, performed incision of the sphincter for no more nor less than 1 cm., so that all the fibres of the sphincter were clearly separated. In some of the animals, more extensive section by double cut, quadrant resection, and even avulsion, was done, yet continence returned by means of a fibrous contracture between the circular muscle-fibres. In each of their cases there was a return of resistance to biliary tract drainage to presphincterotomy levels within 4–6 weeks of operation. Even temporary splinting of the severed and dilated sphincter over a Cattell long-arm tube (Cattell and others, 1957) did not change the ultimate return of sphincter continence. Marshall Bartlett and Nardi (1960) report 12 poor results out of 32 cases in sphincterotomy and transduodenal exploration of the pancreatic duct for relapsing pancreatitis. A further surgical procedure had to be carried out in 7 cases.

There are, moreover, dangers in sphincterotomy. In the hands of the experts, Doubilet and Mulholland (1956), there is a mortality of 5·3 per cent. Howard and his associates (Thompson, Howard, and Vowles,

1957) have recently called attention to the dangers of acute pancreatitis occurring after any biliary tract surgery. In 66 patients with this complication 62 had had a choledochostomy and 17 followed the use of the long-arm tube of Cattell. They state that when the sphincteric region is approached mechanically, either with bougies, dilators, or the scalpel, due regard must be taken of inducing pancreatitis. They give details of 4 of their own cases, all of whom died. Walters (1956) counsels caution in sphincterotomy, and reports 2 cases in which a choledochal pressure of 300 mm. saline, severe pain, and ductal spasm, were recorded 2–3 months after sphincterotomy. Moreover, if the sphincter is divided too deeply there is danger of posterior duodenal perforation and a leak. Blatherwick and Pattison (1954) report 3 fatalities due to pancreatitis following sphincterotomy.

In view, therefore, of the impermanence of sphincterotomy in reducing choledochal pressure and its attendant mortality, it seems reasonable to make an extended trial of choledochoduodenostomy in relapsing pancreatitis. Of course, it is impossible to draw conclusions from 9 cases, and choledochoduodenostomy will not look after those cases where stenosis occurs actually in the pancreatic duct. One further acalculous case, not yet admissible in this series, has had complete cessation of attacks for 11 months after operation following frequent trouble over the past year. A lot of further work on this point is necessary before any conclusion can be reached.

Gambill, Comfort, and Baggenstoss (1948), in considering relapsing pancreatitis, found that cholecystectomy gave relief in 3 out of 5 cases, choledochostomy in 4 out of 7 cases, and choledochoduodenostomy in 6 out of 7 cases. They add that there was complete and immediate freedom from pain in the last group and the remaining case had marked, but delayed, relief from pain. Perhaps he had pancreatic duct stenosis. Their follow-up was over 45 months.

Chronic Pancreatitis.—In 9 cases a diagnosis of chronic pancreatitis was made on the basis of gross thickening of the gland throughout its length. The finding of nodular masses in the head alone was not accepted as indicating chronic pancreatitis, as it is such a common accompaniment of gall-stones.

Of these 9 cases, 5 had stones in the common duct and 4 were reported as having a normal biliary tree. Details of these are:—

G. M., male, aged 74 years. 1957: Jaundice with a grossly enlarged pancreas at operation, a normal biliary tree apart from a dilated but blue common bile-duct. Section showed a chronic pancreatitis. He is now asymptomatic 4 years after choledochoduodenostomy.

E. E., male, aged 69 years. 1954: Had an enlarged thickened pancreas with a purulent cholangitis but no stones could be found. There was stenosis of the sphincter of Oddi. He has been asymptomatic for 7 years since choledochoduodenostomy.

E. M., male, aged 73 years. 1954: Jaundice, fever, grossly enlarged pancreas, no stones. Choledochoduodenostomy performed. He died 2½ years later of a coronary thrombosis. He remained without biliary symptoms up to the time of death.

O. C., female, aged 48 years. 1953: Dilated common duct, stricture of ampulla, no stones, pancreas showed fibrosis throughout. Choledochoduodenostomy performed. She has had no further symptoms in the last 7 years. The stoma was patent in 1955, but the test has not been repeated since.

3. Carcinoma of the Pancreas (10 cases).—Eight are considered and 2 under 'Operative Mortality'.

4. Trauma (5 cases).—

S. B., female, aged 33 years. Following cholecystectomy in September, 1959, developed jaundice: in March, 1960, at laparotomy, stenosis at point of ligation of cystic duct was found, and a choledochoduodenostomy was done. Since this she has had no jaundice, and no rigors, but has had two attacks of pain, each lasting 30 min. The stoma is patent and liver function tests are normal.

E. R., female, aged 28 years. Cholecystectomy, May, 1956: jaundice followed 2 months later. Choledochoduodenostomy for obliteration of duct at region of cystic duct. The patient had a very severe attack of infective hepatitis 2 years later. It is now 3¼ years since operation, and patient remains well, with no biliary symptoms. Whether the severity of the infective hepatitis was due to the choledochoduodenostomy is a problem. The stoma is widely patent to barium and the liver function tests show no abnormality.

E. G., female, aged 48 years. 1950: Cholecystectomy. Following this she had three attacks of jaundice with biliary pain. In June, 1951, choledochoduodenostomy for obliteration of common duct below region of cystic duct. Now, nearly 10 years later, the stoma is patent to barium, the liver function tests show no abnormality, she has no jaundice or pain, but gets occasional shivers in the winter.

R. S., male, aged 52 years. In June, 1957, developed biliary fistula following cholecystectomy for acute cholecystitis. One month later at laparotomy the common duct was found to be strictured and it was repaired over a T-tube. This was satisfactory till 17 months later, when jaundice and rigors occurred. A choledochoduodenostomy was then done, and now, more than 2 years later, he has no symptoms apart from heartburn; the stoma is patent to barium, and his liver function tests show no abnormality.

A. H., female, aged 79 years. Developed a biliary fistula following cholecystectomy and choledocholithotomy for jaundice in August, 1950. Three weeks later, at laparotomy, a stricture was found and a choledochoduodenostomy was carried out. She remained well for 7 years, and died of a coronary thrombosis. She had no biliary symptoms, but her conjunctivæ remained subicteric till her death. No stomal patency or liver function tests were done.

5. Liver Fluke (1 case).—On opening the common duct, multiple *Clonorchis* adult worms were found and a choledochoduodenostomy was done. Stock and Tinckler (1955), writing from Hong Kong, report on 16 cases of cholangio-hepatitis due to liver fluke infestation and stones, and noted a satisfactory response when a choledochoduodenostomy was done. Fung (1961) further reports on a total of 141 cases with a 10 per cent recurrence rate. He states that all these cases showed air in the biliary tract signifying patency of the anastomosis, though not necessarily adequacy of the stoma. He has found sphincteroplasty as described by Jones and Smith (1952) preferable to this, and quotes 16 cases without recurrence of trouble.

6. Post-cholecystectomy Syndrome (1 case).—This case underwent choledochoduodenostomy 1 year after cholecystectomy for stones. Symptoms persisted. He was certainly no better, and apparently no worse, after the choledochoduodenostomy operation. His stoma remains patent and liver function tests are normal. Kjaergaard, of Copenhagen (1953), has recorded benefit in such cases. It must be

difficult to be sure that the pain arises from dyskinesia of the sphincter of Oddi, or even if it did, whether such an operation would be of benefit.

7. Choledochal Cyst (1 case).—This patient, a woman aged 28 years, has done very well over a period of 5 years. Stammers (1961) reports complications following this procedure when, 20 years later, his patient complained of pain. On opening the choledochal cyst he found tomato skins, and converted the anastomosis into a choledochojejunostomy.

8. Post-gastrectomy (1 case).—

A man aged 38 years had a Polya gastrectomy for a duodenal ulcer, with deep penetration of the head of the pancreas, making dissection difficult. Six months later he developed obstructive jaundice and at laparotomy it was found that the common bile-duct was compressed by an inflammatory swelling in the head of the pancreas. A cholecystduodenostomy was done. A few months after this, jaundice recurred, this time with rigors. Five months later a choledochoduodenostomy was done, when a soft stone, looking like a date stone, was removed from the common duct. That was 7 years ago, and he has had no further trouble except that he has to wear a belt for an incisional hernia. The biliary tree is clearly outlined by gas: his liver function tests are within normal limits.

9. Dilated Common Bile-duct (8 cases).—The remaining cases are included under this somewhat indeterminate heading. In each case the most noticeable feature was the dilatation of the common duct, and 5 of the cases had had a previous cholecystectomy for stones. In none of the cases were stones found on choledochostomy, and in all cases there was a variable degree of stenosis of the sphincter of Oddi. In each case, except one, there has been a good response to choledochoduodenostomy. The remaining case reports occasional colic, which is sometimes severe, and is improved by sodium taurocholate, as mentioned above. He has persisted like this over the last 2½ years, and on one occasion had to be admitted to hospital on this account. He has had no jaundice or rigors, and his stoma is patent to barium, with normal liver function tests.

SUMMARY

One hundred and twenty-five cases of choledochoduodenostomy have been studied at an average of 5 years after operation. The patients were assessed with particular regard to recurrence of jaundice, deterioration of liver function, and ascending biliary infection. In 72 patients barium meal examinations were performed to determine the degree of patency of the stoma. In 93 per cent of those examined radiologically, complete patency of the stoma was demonstrated, and these patients were free from complications. It is concluded that, provided a stoma of at least 3 cm. is made, permitting easy entry and egress of intestinal contents, no biliary or hepatic complications will ensue. If the stoma contracts or is inadequately fashioned, food debris may be retained in the common duct and this may result in jaundice and biliary infection. Five such cases are recorded.

In 79 cases bile-duct stones were the indication for the operation, especially when the duct contained 'biliary mud' and there was evidence of stenosis at the lower end.

In 4 cases of acalculous relapsing pancreatitis, complete relief of symptoms has resulted following choledochoduodenostomy, suggesting that the operation is worthy of an extended trial in such cases. The operation is relatively safe.

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INTESTINAL OBSTRUCTION IN CHILDREN DUE TO ASCARIS SIMULATING INTUSSUSCEPTION

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IN tropical regions human infestation with ascaris remains common, especially in children, and the incidence of surgical complications is high. However, in reviewing the literature, a correct diagnosis of this condition is not often made pre-operatively, and patients may be subjected to unnecessary surgical procedures.

FIG. 321.—*Case* 2. Radiograph of abdomen showing wisp-like radiolucent lines arranged in a parallel manner.

Aiken and Dickman (1957) described an X-ray sign of "wisp-like radiolucent lines in a somewhat parallel manner within a more dense shadow representing a mass of ascaris". No mention was made of any specific physical signs.

Opinion varies as regards treatment: Garcia (1956) described 5 cases requiring surgical intervention; Basnuevo (1957) advocated conservative treatment.

In the 4 cases seen recently by the author, some interesting clinical and radiological features were noted and merit reporting, for they give good grounds for correct clinical diagnosis of the condition. The choice of proper treatment becomes easier in the light of a firm diagnosis.

CASE REPORTS

Case 1.—A 4½-year-old girl was admitted through the Casualty Department on March 6, 1961, as a case of intussusception, complaining of sudden onset of abdominal colic 5 hours previously. The attacks of pain occurred at intervals of 10–15 min. The patient had repeated vomiting of ingested food, amounting to 7–8 times, starting one hour after the onset of the pain. No history of passing stool, blood, or mucus per rectum. No feverishness.

EXAMINATION.—The child was in good general condition. The abdomen was slightly distended and soft. A sausage-shaped mass 4 in. × 2 in. was located just to the right of the umbilicus, with its long axis lying transversely. Deep palpation of the mass showed crepitation; the feeling was exactly as in a case of subcutaneous emphysema, suggesting air being trapped in a honeycomb mass. Percussion over the mass was tympanitic. Bowel-sounds were not excessive. P.R. no mass was felt, yellowish stool found on glove-finger, no blood or mucus. Temperature 97 F., W.B.C. 8000/c.mm., Hb 13·5 g. per cent. Plain radiograph of abdomen showed no abnormal fluid levels or distended loops of bowel.

Intussusception was diagnosed and laparotomy done.

AT OPERATION.—The sausage-shaped mass was found to be the dilated terminal ileum heavily infested and obstructed by ascarides. The small intestine proximal to the mass was not significantly dilated. The ascarides could be gently squeezed through the ileocæcal valve into the cæcum. Appendicectomy was done and the wound closed.

Post-operatively patient was given piparazine (antepar) orally. About forty ascarides were passed per rectum on the third post-operative day. Patient was discharged on March 13.

In retrospect, round-worm shadows could be clearly seen in the radiographs coinciding with the position of the palpable mass.

Case 2.—A 6-year-old boy was admitted on May 22, 1961, through Casualty as a case of intussusception, complaining of the sudden onset, 9 hours previously, of attacks of colicky abdominal pain at intervals of about 10 min. There was repeated vomiting of ingested food. No bowel movements since the onset of pain.

ON EXAMINATION.—The patient was in fair general condition and not dehydrated. Abdomen was slightly distended. A sausage-shaped mass size 5 in. × 2 in. was palpable just to the left of the umbilicus, with its long axis lying longitudinally. Deep palpation showed

crepitation and tenderness over the mass. Bowel-sounds not excessive. P.R. no mass felt, normal fæces on glove-finger. Temperature 98° F., W.B.C. 10,000/c.mm., Hb 12·5 g. per cent. Stool on microscopy showed many ascaris ova, no blood, no mucus. Plain radiograph of abdomen showed wisp-like radiolucent lines arranged in a parallel manner within a fluffy soft-tissue shadow in the left lower quadrant, corresponding to the site of the palpable mass (*Fig.* 321).

The pre-operative diagnosis was intussusception, but, in view of the previous experience with *Case* 1, there was a strong suspicion that the case was again one of ascaris causing intestinal obstruction.

At Operation.—A dilated loop of ileum (18 in. from the ileocæcal valve) full of entangled ascarides was located in the left side of the abdomen. Marked mesenteric lymphadenopathy was present. A small ileo-ileal intussusception (1 in. long) was found proximal to the mass of ascaris. The segment involved in the intussusception was viable and easily reduced. The small gut proximal to the mass was not dilated. Attempts to push the ascarides through the ileocæcal valve failed. Enterotomy was done in the proximal normal part of the small intestine. The chosen loop was packed off from the other structures and a longitudinal incision was made at the antemesenteric border. The ascarides were removed by using long forceps. The enterotomy was closed transversely in two layers with thread. Appendicectomy was carried out and the wound closed without drainage.

Following the operation the patient was given intramuscular penicillin and streptomycin with aspiration through a Ryle's tube and intravenous fluid. On the second post-operative day the patient tolerated fluid diet well. Antepar was given. The patient was discharged on May 30 without complications.

Case 3.—A boy, aged 6 years, was admitted on May 29, 1961, through Casualty as a case of intussusception, complaining of repeated episodes of abdominal colic and vomiting for 10 hours. Patient passed a few ascarides at age of 3 years.

On Examination.—Fair general condition. Abdomen was distended with ladder patterns. A vague tender sausage-shaped mass was palpable just to the left of the umbilicus, the long axis being parallel to the left inguinal ligament. Crepitation was palpable and percussion was tympanitic over the mass. Bowel-sounds were hyperactive. P.R. normal stool was recovered from the gloved finger showing very many ascaris ova but no blood or mucus. Temperature 100° F., W.B.C. 11,000/c.mm., Hb 12 g. per cent.

Radiography of the abdomen showed dilated loops of small intestine without abnormal fluid levels. Multiple shadows of ascarides were seen in a dilated loop of small bowel at a site corresponding to the clinically palpable mass (*Fig.* 322).

Diagnosis of intestinal obstruction due to ascaris was made and the patient was treated conservatively with aspiration through a Ryle's tube and intravenous fluids. Antepar was given orally. The patient could tolerate fluid diet well after 8 hours. On the third day after admission the patient passed about 80 ascarides per rectum; all the abdominal signs and symptoms disappeared, and he was discharged well. Repeat radiography showed the shadow of 1 ascaris instead of the initial multiplicity.

Case 4.—A boy of 4 years old was admitted through Casualty on Aug. 4, 1961, as a case of intussusception. He had acute onset 8 hours previously of repeated episodes of severe abdominal colic at 10–20-min. intervals. He vomited ingested food twice. During the attacks the child turned pale and doubled himself up. He had no bowel movement on the day of admission. No feverishness. The patient previously was in good health except that he vomited some ascarides 2 weeks ago.

On Examination.—He was in good general condition. Temperature 98° F. Not dehydrated. Inspection showed visible peristalsis in the centre of the abdomen. A large horseshoe-shaped mass size 5 in. to 2½ in. was located in the right side of the abdomen just lateral to the umbilicus. Palpation showed crepitation and some cord-like structures within the mass. Percussion over the mass was tympanitic. Bowel-sounds were moderately increased. P.R. no mass felt. Normal-coloured stool found on gloved finger showing many ascaris ova on microscopy. Hb 12·5 g. per cent, W.B.C. 10,400/c.mm.

Fig. 322.—*Case* 3. Multiple coiled ascarides shown in left iliac fossa corresponding to the palpable mass.

Radiography of the abdomen showed round-worm shadows in the right side of the abdomen corresponding to the site where the mass was felt.

Diagnosis of ascaris causing intestinal obstruction was made and the patient was treated conservatively as outlined in *Case* 3. The mass could no longer be palpated on the second day after admission, and on the third day he passed about 60 ascarides per rectum. He was discharged well on Aug. 10, 1961, having tolerated full diet for two days.

DISCUSSION

Diagnostic clinical and radiological features are present in all these 4 cases. Relevant treatment may be easily chosen when diagnosis is reasonably assured. These are discussed separately:—

1. Age-group.—According to Aird (1957), nearly 80 per cent of all intussusceptions occur in infants during the first year in life. In the 4 cases reported the range of age varied from 4 years to 6 years. In Aiken's series all except one were over 2 years. It may be suggested that in endemic areas a child older than 2 years presenting with the clinical symptoms of intussusception should be viewed with suspicion.

2. History.—The character of pain and vomiting was indistinguishable from intussusception. However, the characteristic passage of mucus and blood in some form per rectum in cases of intussusception

was absent in all our cases. Even on microscopical examination, blood and mucus could not be demonstrated, which should be expected in view of the nature of the pathology.

3. Physical Signs.—General condition of the patients was disproportionally good considering the large size of the masses and the number of hours which had elapsed before they were seen. So was the mild degree of abdominal distension in *Cases* 1, 2, 4. Gas and fluid in small amounts could probably still go through the clefts among the ascarides and the obstruction was therefore incomplete.

Pyrexia was absent in *Cases* 1, 2, 4, and mild in *Case* 3, in contrast to the invariable rise of temperature in cases of intussusception (Morrison and Court, 1948).

The abdominal masses were always located more centrally than they would be if the cases were intussusception of the common ileocolic type (*Figs.* 321, 322). This is probably due to the fact that the masses of ascarides are always in the small intestines and the pathology does not involve the colon.

Crepitation when demonstrated is pathognomonic. It is easy to imagine how a large mass of entangled ascarides producing a honeycombed structure and trapping among them the gas in the distended bowel, can give this sign. This same reason explains why a tympanitic note is found on percussion of the mass instead of a dull percussion note in cases of intussusception.

The absence of blood and mucus from the stool specimens and the presence of a large number of ascaris ova should be further indicative of the diagnosis.

4. X-ray Features.—The author has observed and fully agrees with the finding of Aiken and Dickman (1957), namely, the presence of wisp-like radiolucent lines in a somewhat parallel arrangement within a more dense shadow. The fluffiness of the shadow may be due to movement of the ascarides during the radiographic examination. In some cases the outline of multiple ascarides is clearly visible if looked for (*Fig.* 322). These two findings are diagnostic, especially if they are located in the radiographs in a position corresponding to the clinically palpable mass. Barium enema, when attempted, should show completely normal colon and rectum, as the ascarides usually cause obstruction proximal to the ileocæcal valve. It was not carried out in the 4 cases reported.

5. Treatment.—

a. Conservative.—When a correct diagnosis is made and in the absence of obvious signs of such complications as volvulus, perforation, or peritonitis, conservative treatment is the method of choice. The patient is put on gastro-intestinal suction and intravenous fluid. Antepar is administered orally or through a Ryle's tube. Careful and frequent assessment of the progress of the patient is made. Vomiting usually stops, aspiration steadily diminishes, and the patient can tolerate oral fluid soon, followed by passage of the ascarides per rectum.

b. Surgical Intervention.—

i. When the abdomen is opened in cases of impending or actual complication; or, in misdiagnosed cases, attempts should be made to manipulate the ascarides through the ileocæcal valve.

ii. Enterotomy and evacuation of the ascarides is justifiable when manipulation fails. It is argued that enterotomy is dangerous, for the distended and œdematous ileal loop containing the ascarides when opened may fail to hold stitches, and ascarides may burrow through the suture line to the general peritoneal cavity. The author considers it safe provided enterotomy is done on the normal bowel proximal to the dilated loop, and the use of non-absorbable suture material together with a careful suturing technique avoids the danger of perforation. The latter is further minimized by early post-operative treatment with antepar, which paralyses the remaining ascarides, if any.

iii. Resection and ileo-ileostomy should be reserved for cases where gangrene or perforation has complicated the issue. Provided careful technique is exercised, primary re-anastomosis after resection should be a safe and simple procedure. Resection with double ileostomy and secondary closure, advised by Aiken and Dickman (1957), runs the danger of wound infection and difficulty of adjusting fluid and electrolyte balance, as well as the necessity of a second operation, and is probably unnecessary.

SUMMARY

1. Four cases of infestation with ascaris producing intestinal obstruction and mimicking intussusception are reported. The diagnosis was made correctly in the third and fourth cases.

2. A new clinical sign—namely, crepitation in a palpable tympanitic mass—is described, and this, together with other clinical features and previously reported radiological signs, should make accurate diagnosis possible.

3. Conservative treatment is both justifiable and successful in the absence of complications, but if a laparotomy is carried out manipulation of the ascarides through the ileocæcal valve, enterotomy, and evacuation or, as a last resort, resection and anastomosis, may be required.

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INTERMITTENT SUBCLAVIAN VEIN OBSTRUCTION

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SPONTANEOUS occlusion of the axillary or subclavian vein, often referred to as 'effort thrombosis', is a well-known clinical entity. The nomenclature of the condition does not seem to be entirely satisfactory. In a review of 320 cases, Hughes (1949) found that among those cases where the onset was described, in 13 per cent symptoms came on gradually or during sleep, with no history of trauma or 'effort'.

Furthermore, it would appear that exploration of the vein has not always yielded evidence of thrombosis (Wulsten, 1931b; Puhl, 1937; Hauge, 1942), although undoubtedly thrombosis was present in most cases. In the case reported below venographic examinations performed on two occasions showed no evidence of axillary vein thrombosis, and this fact will be commented on in more detail later.

The condition was first described by Paget in 1875 and later by Schrotter in 1884. Hughes (1948) suggested that it should be called the 'Paget–Schrotter syndrome'. It occurs predominantly in males during the third and fourth decades, and the right arm is affected more often than the left. The onset may be quite sudden, but it usually requires 12–48 hr. to develop, and some cases have premonitory symptoms for some days. When there is a history of effort or trauma it is often of a mild variety. Many types of activity have been cited as precipitating causes, from painting walls and ceilings, lifting objects off high shelves (Ffrench, 1944; Townsend, 1952) to spanking a child (Barnett and Levitt, 1951). It may be significant that in many cases elevation of the limb seems to have been a factor.

Swelling of the limb is usually first noticed by the patient, but pain, often of a dull aching or bursting character, may occur at the onset. The arm and shoulder become involved in swelling which does not pit on pressure. Superficial distended veins over the shoulder and anterior aspect of the upper chest are seen. Tenderness and induration along the line of brachial, axillary, or subclavian veins may be present. A bluish discoloration of the limb is usual and there may be paræsthesia or pruritus.

Some reports give the impression that, following a sudden onset, the acute condition will subside rapidly, particularly if anticoagulants are employed, and that sequelæ or recurrences are unusual. However, Aird (1957) states that many patients are left with a sense of heaviness in the arm and Hughes (1948, 1949), Matas (1934), Veal (1940), and Roelsen (1939, 1944) draw attention to the fact that persistent symptoms are not uncommon. Kleinsasser (1949) found that out of 56 cases only 13 had no residual symptoms. Both Hughes and Kleinsasser found that patients may have to change their occupation because of persisting disability.

Pulmonary embolism is surprisingly rare in this condition, although it may occur (Barnett and Levitt, 1951; Samuels, 1951). No case of fatal pulmonary embolism has been reported. The fatal case of Moure (1914), where axillary vein thrombosis was associated with a fracture of the humeral neck and treated by massage, hardly comes into the category under consideration.

The following case report is of interest from the aetiologic, symptomatic, and therapeutic aspects.

CASE REPORT

In July, 1958, Mrs. O. S., aged 37 years, a married woman with four children, began having attacks of blueness and swelling in her left arm. She was unable to recall the precise time and mode of onset, but observed that certain kinds of activity would initiate an attack. At first the arm remained considerably swollen between attacks. The attacks were accompanied by a dull aching pain in the left shoulder and upper arm. The types of activity precipitating an attack were wringing out clothes by hand, knitting, or washing the right side of her body with the left hand. When she was first seen in November, 1958 (four months after the onset), she was still having these attacks, but she thought that the residual swelling of the arm between attacks was less. She found the symptoms distressing and they interfered with her housework.

Her previous health had been good, but she had noticed a swelling in the lower part of her neck in the midline anteriorly. This had been present for six years, was painless, and enlarging slowly.

Physical examination revealed a rather obese but healthy-looking, cheerful woman. At rest the left arm had a slightly purplish colour, and above the elbow it was swollen when compared with the other side. Ten centimetres above the olecranon the difference in circumference between the two sides was 3·5 cm. There was no difference in skin temperature or moisture between the two sides. Blood-pressure was 130/75 on both sides. There were prominent veins over the left upper arm, extending across the shoulder and anterior axillary fold to the left pectoral region. No abnormality was felt in the axilla or supraclavicular fossa. There was no tenderness along the line of the main vessels. The radial pulse was not affected by traction on the arm in various positions. Immersion of the hand in warm or cold water produced no effect.

By performing any of the precipitating exercises mentioned above, she could readily reproduce an attack of blueness and firm swelling of the arm which took 1–2 min. to develop fully. After cessation of the exercise the arm would return to its former state within 5–10 min.

She had a nodule on the lower pole of the left lobe of the thyroid gland. The remainder of the physical examination was unremarkable.

X-ray examination of the thoracic inlet and chest showed slight displacement of the trachea to the right by the thyroid nodule (*Fig.* 323).

A venogram was performed on the left arm. This showed a patent axillary vein, though no filling of the venæ comitantes of the brachial artery was demonstrated. However, the examination was considered unsatisfactory.

Radio-active iodine studies revealed normal thyroid uptake and a 'cold' nodule in the left lobe. Excision of the nodule was advised, and she was admitted to hospital. In

hospital the left arm appeared to improve with elastic bandaging and elevation. The thyroid nodule proved to be a benign follicular adenoma. On discharge from hospital the left arm was much improved.

She was seen again nine months later, with a history of continuing attacks of swelling, blueness, and pain in her left arm. Apparently the condition relapsed on her return to normal activities, and she felt that the attacks were becoming more frequent and easier to produce by minor exertion. She had also experienced a similar aching pain in the right shoulder, but no other symptoms had developed in the right upper limb. On this occasion the clinical findings in the limbs were essentially as before, there being a difference in circumference of 2 cm. between the upper arms at rest.

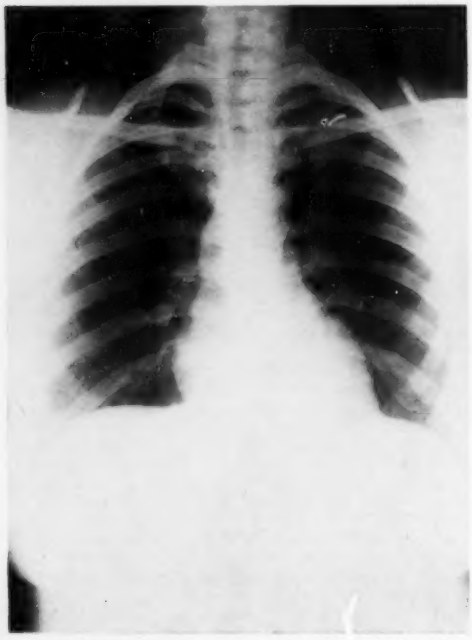

FIG. 323.—Pre-operative chest film showing slight deviation of the trachea to the right due to a thyroid nodule. No abnormality in bony thoracic inlet.

A second venographic examination was undertaken and this time a cardiac catheter was passed up the basilic vein with the object of obtaining precise information about the nature and level of any venous obstruction. The catheter passed with ease into the superior vena cava, and during withdrawal 50 per cent 'hypaque' was injected at intervals under direct fluoroscopic observation. There appeared to be no mechanical block along the line of the brachial, axillary, or subclavian veins. However, in hind sight with knowledge of the operative findings, we think that one of the films showed obstruction at the point where the vein passed over the first rib (*Fig.* 324). Cervicodorsal sympathetic block was performed and following this it was not possible to induce an attack until the effects of the block had passed off.

It was felt that some obstructive or irritative lesion affecting the veins at the thoracic inlet, the nature of which was uncertain, must be present. It was therefore decided to explore the thoracic inlet and perform upper dorsal sympathectomy.

AT OPERATION.—An anterior supraclavicular approach was employed. In the course of the dissection a small nerve-trunk was found lying anterior to the subclavian vein. This was thought to be the nerve to subclavius, but further dissection showed that it was a branch from this nerve which was passing downwards in front of the vein to enter the thorax between the vein and the upper border of the first rib. Stimulation of this nerve produced a diaphragmatic contraction and it was obviously an accessory phrenic nerve. It was noted that tension on the nerve kinked and obstructed the subclavian vein, and this could also occur with the shoulder girdle in certain positions when the nerve to subclavius and accessory phrenic nerves were pulled taut. Speculation at the time suggested that this was, in fact, the cause of the venous obstruction. Further exploration revealed a normal-looking phrenic nerve lying on scalenus anterior, which on stimulation produced normal diaphragmatic contraction. It was decided that the accessory nerve could be safely sacrificed. Upper dorsal sympathectomy was then performed as planned. The anatomical findings are represented semidiagrammatically in *Fig.* 325.

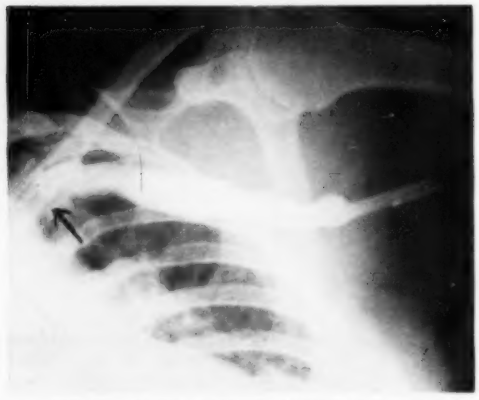

FIG. 324.—Second venogram, showing 'cut-off' of subclavian vein due to occlusion by accessory phrenic nerve. This feature was only shown on one of the films taken during this examination.

POST-OPERATIVE COURSE.—Apart from a mild superficial wound infection the patient made a straightforward post-operative recovery. The left arm and hand were warm and dry, and no Horner's syndrome was present. Before leaving hospital the patient was unable to reproduce her previous symptoms and she seemed to be well pleased with the result.

She was reviewed a month after discharge and she had remained free of symptoms attributable to venous obstruction in the left upper limb. She noticed dryness of the left hand and a slight tendency to increased perspiration from the right arm and hand. Eight months later she was symptom-free, and no dilated veins were seen in the left upper arm and shoulder region.

DISCUSSION

We feel that the cause of this patient's symptoms was compression of the subclavian vein by a prevenous accessory phrenic nerve.

It does not seem to be widely appreciated that the phrenic nerve is subjected to many variations. Normally both the phrenic nerve and accessory phrenic (when present) pass into the thoracic cavity between the subclavian vein and artery. The variations which may occur have been well described by

the older anatomists, and seen to be periodically rediscovered. Quain (1909) describes the following variations:—

1. Accessory roots may be received from C.2, C.6, descendens cervicalis or ansa hypoglossi. The nerve to subclavius may contribute a filament and occasionally the greater part of the nerve arises from this source.

2. The phrenic nerve may pass in front of or behind the subclavian vein.

3. An accessory phrenic nerve may arise from C.5 root or from the nerve to subclavius. This may pass either in front of or behind the subclavian vein to enter the thorax, where it joins the main nerve.

4. Occasionally the phrenic nerve may pass through the subclavian vein, being excluded from the lumen by an endothelial tube, or the vein may make a ring around the nerve. Such a case was reported by Zeren in 1952.

Other anomalies, particularly at the roots of origin, have been described by Köle (1953).

In excellent reviews of the problem of venous obstruction of the upper extremity, Hughes (1948, 1949) critically examines the various theories regarding the aetiology of this condition. Compression of the vein by bony, muscular, or ligamentous structures in the axillary region are considered. On the basis of his own anatomical observations he draws attention to the possibility that a prevenous phrenic or accessory phrenic nerve may cause subclavian vein occlusion, particularly during inspiration when the diaphragm descends, straightening the course of the nerve.

In our own patient we suggest that when the shoulder girdle was in certain positions tension on the nerve to subclavius transmitted to the accessory phrenic nerve caused kinking of the subclavian vein and intermittent venous obstruction.

We believe that this is the first case reported where definite evidence of venous obstruction due to this cause has been found at operation and the condition has been relieved by division of the abnormal nerve.

There is no evidence in this case that there was ever thrombosis in the axillary or subclavian veins. Although the first venogram showed no filling of the brachial vein or the venal comitantes of the brachial artery, clinically there was no evidence of thrombosis along this easily palpable portion of the venous system.

One of the films taken during the second venographic examination showed a neat linear 'cut-off' where the subclavian vein crosses the first rib (*Fig.* 325). However, the significance of the 'cut-off' was not appreciated. In the light of the operative findings we suggest that the 'cut-off' was due to compression of the vein by the accessory phrenic nerve. We feel that the intermittent and chronic nature of this patient's symptoms are more plausibly explained by some recurring mechanical obstruction to the subclavian vein.

It may be objected that sympathectomy would in any case cause symptomatic relief. Cervicodorsal sympathetic block has been shown to give good symptomatic relief in this condition (DeBakey, Ochsner, and Smith, 1942). Release of vasomotor tone appears to encourage the dilatation of venous collaterals. Since operation our own patient has lost her superficial venous collaterals, and these were prominent before operation, even when the main veins were shown on venography to be patent. Unless some obstructing factor has been remedied one would expect enlarged venous collaterals to persist with or without sympathectomy. We think that sympathectomy was performed unnecessarily on this patient.

Management.—It is appreciated that we are dealing with a condition for which one or more of several factors may be responsible. It is probably because of this that surgical opinion appears to be rather uncertain on the approach to this problem. On the whole, most surgeons would advise against operative interference. Certainly, operation should only be considered in cases with troublesome or disabling persisting symptoms.

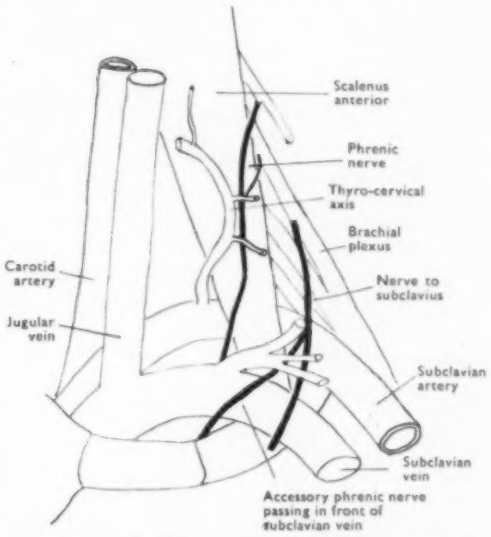

FIG. 325.—Semidiagrammatic representation of operation findings. Normal phrenic nerve is shown. Accessory phrenic coming off nerve to subclavius is shown passing in front of the subclavian vein, then entering the thorax.

Whenever 'effort' thrombosis is seen in the early or acute phase it would seem reasonable to commence treatment with anticoagulants without delay. If thrombosis is present, extension of the thrombus will be prevented, thus limiting residual impairment to the venous drainage from the limb. If no thrombus has formed, and the cause of the obstruction is a mechanical factor, as in the case described above, properly controlled anticoagulant therapy will do no harm and may prevent thrombosis occurring. No active intervention is indicated at this stage, though if marked venous engorgement of the limb and pain are present cervicodorsal sympathetic block may be performed and repeated if necessary.

It is possible that fibrinolytic agents now available (Actase—Ortho Pharmaceuticals; Thrombolysin—Merck, Sharp, & Dohme) may be of value in the early stages of this condition. Such treatment should be initiated as soon as possible after the onset, for after

five to seven days intravenous thrombi do not appear to be susceptible to lysis (Chapple and Singher, 1959). The question of the combined use of fibrinolytic agents and anticoagulants in the treatment of venous thrombosis does not yet appear to be settled, but it would seem to be a rational procedure (Moser, 1958).

Among those patients first seen at a later stage, at the time when a thrombus (if present) will have undergone partial or complete organization, and having significant persisting symptoms, we would stress the importance of distinguishing cases with thrombus in the vein from those with extravenous obstruction such as was found in our own case. For this, careful venographic studies should be done. If no thrombus is demonstrated, it may be possible to form some opinion as to the site and nature of the obstruction. This will entail examination with the limb in various positions. In our own case, not knowing what to look for, we believe we failed to recognize the significance of the X-ray appearances shown in *Fig.* 324.

If a mechanical obstruction other than thrombosis is demonstrated, the feasibility of surgical relief should be considered. We suggest that the cause in most cases will be a prevenous phrenic or accessory phrenic nerve, and that if this condition becomes more widely recognized more examples will be found.

When a prevenous accessory phrenic nerve is encountered there can be no objection to sacrificing it if a normal main phrenic nerve can be demonstrated. If all the phrenic fibres are prevenous, it has been suggested (Hughes, 1948) that phrenic crush may relieve pressure on the vein by diminishing diaphragmatic movement and consequent tugging on the nerve. Hughes also suggests that in a chronic case some form of venous by-pass could be performed. We can find no report of either of these procedures being successfully performed. The continued patency of any form of venous by-pass or reconstruction would be doubtful, and incidental damage to collaterals might be to the patient's disadvantage. When the obstruction is due to a prevenous phrenic nerve, scalenotomy (McLaughlin and Popma, 1939) with mobilization of the phrenic nerve might offer a prospect of relief providing the subclavian vein is not thrombosed.

Another surgical procedure recommended in the literature is phlebotomy with removal of the clot (Bazy, 1926; Biebl, 1939). To be effective this would have to be done in the acute stage. The exposure of the subclavian vein is not without technical difficulties and valuable collaterals may be damaged. As many subjects will in any case have negligible residual symptoms, this procedure does not recommend itself. Venolysis (Wulsten, 1931a), excision of the thrombosed segment (Delbet, 1913), and perivenous and periarterial sympathectomy (Roelsen, 1939) have been described. However, if the vein is thrombosed, upper limb sympathectomy probably offers the best prospect of symptomatic relief by encouraging dilatation of venous collaterals.

SUMMARY

1. 'Effort' thrombosis of the proximal upper limb veins is not always associated with effort, and symptoms of venous obstruction do not necessarily mean the occurrence of thrombosis.

2. The symptomatology and findings in this condition are briefly reviewed.

3. A case of chronic intermittent venous obstruction of the upper limb due to a prevenous accessory phrenic nerve treated by division of the nerve and upper dorsal sympathectomy is described.

4. Anatomical variations of the phrenic nerve and accessory phrenic nerve are briefly described.

5. The management of the acute and chronic stages of this condition is discussed. In the latter, emphasis is placed on detecting those cases without venous thrombosis. In the acute phase, anticoagulants, sympathetic block, and possibly fibrinolytic agents may be employed. Cases with persistent symptoms but no venous thrombosis may have a prevenous phrenic or accessory phrenic nerve. An accessory phrenic nerve may be divided; otherwise scalenotomy and mobilization of the phrenic nerve may be performed.

When thrombosis is present, upper dorsal sympathectomy probably offers good relief for persistent symptoms of venous obstruction.

6. Other surgical procedures previously advocated are briefly mentioned.

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ŒSOPHAGEAL STRICTURE AFTER PARTIAL GASTRECTOMY

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THE complications of gastrectomy for peptic ulcer are various and well documented, for the follow-up of such patients is usually very careful. Œsophageal stricture after partial gastrectomy occurs rarely, but in the reported instances it is severe and rapid in development.

Three further cases (curiously occurring within three months) are reported, and the possible mechanisms discussed.

Case 1.—Mr. F. W., a 70-year-old butcher with a long history of peptic ulcer pain and three previous transfusions for melæna, perforated his duodenal ulcer on May

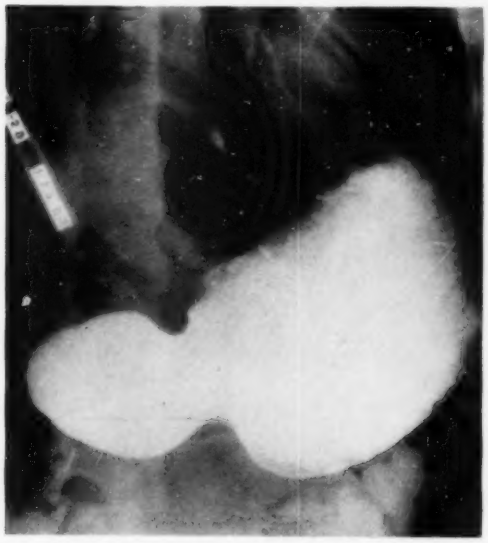

FIG. 326.—Photograph of barium meal in *Case* 1 on Aug. 13, 1959. Gastric distension from duodenal obstruction is seen. No abnormality of the œsophagus was detected in this series of films.

30, 1959, and this perforation was oversewn. Duodenal obstruction followed with persistent vomiting, epigastric colic relieved by vomiting, weight-loss, dehydration, and anæmia.

Succussion splash and visible peristalsis were observed in the epigastrium. Barium meal examination on admission on Aug. 13, 1959 (*Fig.* 326), confirmed the diagnosis of duodenal obstruction. No abnormality of the œsophagus was apparent in these films.

On Aug. 17, partial gastrectomy with antecolic gastrojejunostomy was performed for penetrating duodenal ulcer. On Aug. 20 he complained of a sore throat, and the Ryle's tube, which had been in place since operation, was removed. On Aug. 25 he developed a small duodenal fistula and this closed spontaneously 8 days later.

On Sept. 1, 15 days after operation, he complained of food sticking in his throat, and of the immediate vomiting of any food taken. Barium swallow examination on Sept. 4 (*Fig.* 327) demonstrated a stricture of the distal œsophagus. Self-bouginage was begun, but a further barium swallow on Sept. 24 (*Fig.* 328) showed the stricture to be longer.

Histamine test-meal at this time produced a volume of 20 ml. containing bile in all specimens, no free hydrochloric acid, and a total acidity of 16 units (number of ml. of *N*/10 NaOH to bring the *p*H to 7).

Dilatation through the œsophagoscope to size 32 bougie was performed on the stricture which began 32 cm.

FIG. 327.—Photograph of barium swallow in *Case* 1 on Sept. 4, 1959, 18 days after gastrectomy. Stricture of the terminal œsophagus is seen.

from the incisor teeth. He was discharged to continue self-bouginage. Re-admission was necessary on Nov. 24, for dysphagia. Barium swallow (*Fig.* 329) showed the stricture extending from the carina to 2 cm. above the diaphragm, and failure of a swallowed bougie to pass the stricture. Œsophagoscopy and dilatation again relieved the dysphagia, and *Figs.* 330, 331, photographed through the œsophagoscope, show the upper end of the stricture of the œsophagus with reflux through the stricture and œsophagitis. *Fig.* 332 shows the most recent swallow in which improvement has occurred with self-bouginage.

He was last seen on Sept. 14 and at this time he was swallowing a Hirst bougie daily, and eating minced food without dysphagia. No hiatus hernia could be demonstrated radiologically, but free gastro-œsophageal reflux was seen at œsophagoscopy.

Case 2.—Mr. R. V., a 64-year-old dockworker, was well, apart from occasional belching, until April 25, 1959, when his duodenal ulcer perforated and was oversewn. Three months later retrosternal pain and burning began,

occurring on bending and exertion and after meals. On Sept. 20 he vomited blood, and partial gastrectomy with antecolic gastrojejunostomy was performed.

His convalescence was uneventful until 20 days later, when dysphagia began. In the next two months he lost 4 stone in weight, and had bouts of coughing and dyspnœa after feeding. By the time of admission on Dec. 16 he was able to swallow only fluids. Barium swallow showed complete obstruction from a stricture of the terminal œsophagus (*Fig.* 333). Œsophagoscopy on Jan. 4 demonstrated œsophageal spasm from 31 cm., and a tough fibrous stricture from 39 cm. The stricture was dilated. Later that day he was returned to theatre and the clinical diagnosis of œsophageal perforation was confirmed. The perforation was extended and closed transversely, and a small sliding hiatus hernia was repaired. Seven days later cardiac arrest occurred during bronchoscopy for excessive secretions. The heart was re-started by cardiac massage, but the patient died later the same day. Autopsy confirmed the findings at operation.

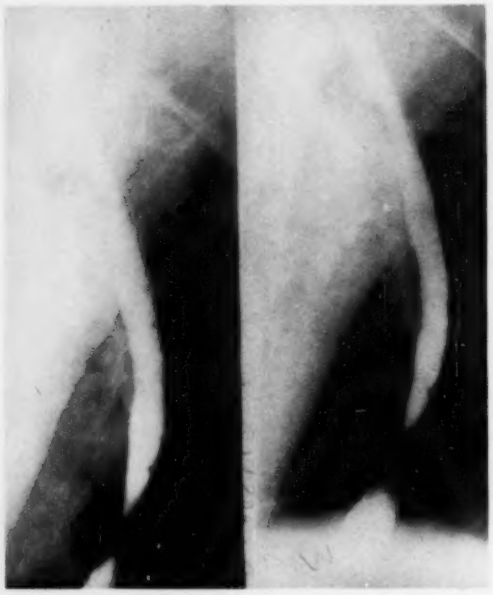

FIG. 328.—Photograph of barium swallow in *Case* 1 on Sept. 24, 1959, 5 weeks after gastrectomy. Compared with *Fig.* 327 the œsophageal stricture is longer and narrower.

FIG. 329.—Photograph of barium swallow in *Case* 1 on Nov. 30, 1959, 3 months after gastrectomy. The œsophageal stricture has extended proximally.

Case 3.—Mrs. T. M., a 59-year-old housewife, underwent laparotomy in May, 1959, because of ten years of periodic post-prandial epigastric pain. A duodenal ulcer was apparently found, and the abdomen was closed with drainage. For the next five months she had increasing vomiting and increasing epigastric pain radiating to the back.

On admission on Oct. 29, 1959, she had gross pyloric stenosis with succussion splash. On Nov. 3 partial gastrectomy with antecolic gastrojejunostomy was performed for penetrating duodenal ulcer with stenosis.

Seven days later dysphagia began for the first time, and barium swallow 3 weeks after gastrectomy demonstrated an œsophageal stricture with ulceration above a sliding hiatus hernia (*Figs.* 334, 335).

On Jan. 6, 1960, the hiatus hernia was repaired from above with transgastric dilatation of the tough fibrous stricture. *Fig.* 336 shows the barium swallow after dilatation at operation.

On Jan. 21 the œsophagus obstructed with food; at œsophagoscopy gross œsophagitis was still present, and the œsophageal stricture was dilated. Regular self-bouginage since then has maintained adequate swallowing.

The common features in these 3 patients are:—

1. Duodenal ulcer. Two had previously perforated, and 2 had duodenal stenosis.
2. Partial gastrectomy with Polya reconstruction (each by a different surgeon).
3. Complete absence of obstructive dysphagia before gastrectomy.
4. Complete dysphagia for solids within 20 days of gastrectomy.
5. Severe œsophagitis revealed by œsophagoscopy.
6. Rapidly developing fibrous œsophageal stricture.

Sliding hiatus hernia was demonstrated in 2 patients, and free gastro-œsophageal reflux was seen at œsophagoscopy in the other.

DISCUSSION

Œsophageal stricture after uncomplicated partial gastrectomy has been reported by Straus (1950), Douglas (1956), and McKeown (1958). Œsophageal stricture after partial gastrectomy with complications (to be discussed later) has been reported by Benedict and Daland (1938) and Craighead (1954). Œsophageal stricture after gastrojejunostomy has been reported by Klein and Hochbaum (1939), Larson, Layne, and Howard (1942), Paul (1943), Bergquist (1946), Allison (1948), Wangensteen and Leven (1949), Douglas (1956), and Bingham (1958).

It would appear that these patients have a common mechanism of reflux of alkaline tryptic jejunal contents into the œsophagus and are comparable with the alkaline œsophagitis and stricture seen after loop œsophagojejunostomy. However, we must first examine what other evidence is available on the mechanism of œsophagitis in similar circumstances.

Mechanisms of Œsophagitis.—Œsophagitis and œsophageal stricture are seen in various circumstances and it is often difficult to define the precise significance to be attributed to one or other factor present.

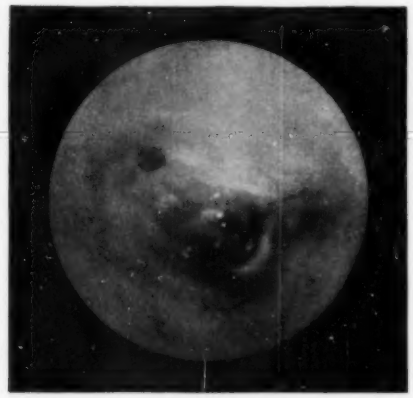

FIG. 330.—Colour photograph of the upper end of the œsophageal stricture in *Case* 1. This photograph demonstrates the stricture, reflux through the stricture, and œsophagitis above that level. (*Single frame from cinéphotograph through the œsophagoscope by Dr. G. Berci, of the University of Melbourne, Department of Surgery.*)

FIG. 331.—Colour photograph through the œsophagoscope of the upper end of the œsophageal stricture in *Case* 1, demonstrating the narrowness of the stricture and the œsophagitis above it.

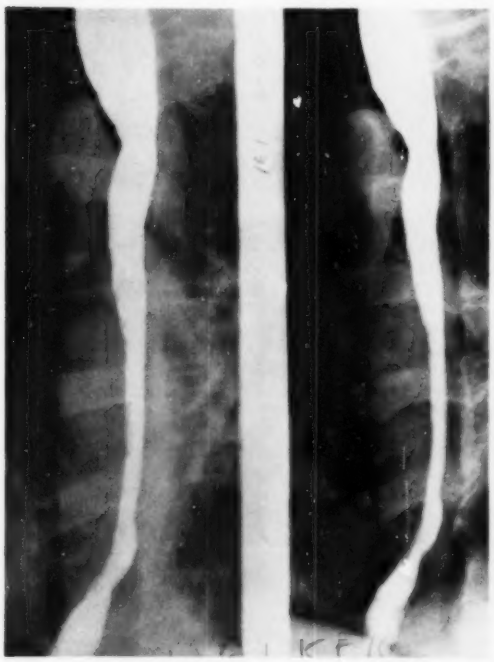

FIG. 332.—Photograph of barium swallow in *Case* 1 on April 27, 1960. Daily self-bouginage has dilated the stricture.

FIG. 333.—Photograph of barium swallow of *Case* 2 on Dec. 14, 1959, 11 weeks after gastrectomy. No barium passed the stricture into the stomach.

The regurgitation of acid pepsin is widely accepted as a common effector mechanism in most of the categories to be discussed, using the analogy of

duodenal ulcer. The part possibly played by other factors is less certain, but the following groups show the contexts in which œsophagitis and stricture are seen.

FIG. 334.—Photograph of barium swallow of *Case* 3 on Nov. 24, 1959, 3 weeks after gastrectomy. Hiatus hernia and œsophageal stricture with ulceration are seen.

FIG. 336.—Photograph of barium swallow of *Case* 3 on Jan. 20, 1960, 2 weeks after repair of hiatus hernia with trans-gastric dilatation of the stricture.

1. *Incompetence at the Gastro-œsophageal Junction.*—The commonest cause of similar pathology is regurgitation of acid pepsin in sliding hiatus hernia. This has been well documented (e.g., Chevalier Jackson, 1929; Allison, 1948; Benedict and Sweet, 1948) and is supported by the evidence of Barrett and Franklin (1949) on the effects of operations for cardio-spasm which render the gastro-œsophageal junction incompetent.

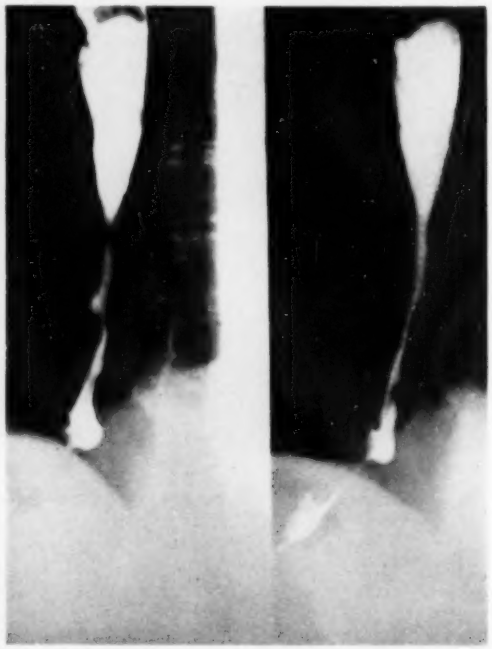

FIG. 335.—Photograph of barium swallow of *Case* 3 on Nov. 24, 1959, 3 weeks after gastrectomy. Œsophageal stricture with ulceration is seen.

2. *Duodenal Obstruction.* — Such acid-peptic œsophagitis is the most probable mechanism in the œsophageal strictures seen when duodenal stenosis occurs after perforated duodenal ulcer is oversewn, as described by Jackson (1932), Vinson (1941), Paul (1943), Bergquist (1946), Franklin (1954), and MacLean and Wangensteen (1956). This is supported by the experimental evidence of Selye (1938), who readily produced hæmorrhagic œsophagitis in dogs by pyloric ligation.

3. *Vomiting.*—Œsophagitis and stricture have been reported in hyperemesis gravidarum and other causes of vomiting (Trotter, 1850; Bartels, 1935; Vinson and Butt, 1936; Bloch, 1940; Vinson, 1941; Palmer, 1952; Mason and Ausband, 1952) and many of the cases of œsophagitis reported after operation have vomiting as a common factor.

It is not clear whether inundation of the œsophagus with acid pepsin is usually responsible (as occurs in pyloric stenosis), or inundation with alkaline-tryptic duodenal contents, for the two are usually inseparable clinically. Alkaline duodenal contents can certainly cause œsophagitis, and this will be discussed later.

That mechanical factors and pressure differences are significant in the production of œsophageal lesions after vomiting is seen in cases of rupture of the œsophagus after vomiting (e.g., Boerhaave, 1724; Weiss and Mallory, 1932).

4. *Indwelling Tube.*—The part played by an indwelling nasogastric tube is less certain. Its presence is a regular finding in case reports of œsophagitis after operation (Bartels, 1935; Bloch, 1940; Benedict, 1946; Mason and Ausband, 1952; Douglas, 1956), and in case reports of œsophagitis after intubation without operation (Bartels, 1935; Vinson and Butt, 1936; Bloch, 1940; Vinson, 1941). Wangensteen, Rea, Smith, and Schwyzer (1939), however, have stated that they have seen no ill effects from over 5000 intubations. In those reported cases other factors were present simultaneously and the time lag between intubation and stricture was often very great. The indication for intubation was usually vomiting, and many of the patients were old and debilitated. The two or three possible mechanisms are not clinically separable.

The tube can facilitate regurgitation of gastric contents, especially with the patient horizontal, by holding open the gastro-œsophageal junction. But the œsophagitis is often severe and progressive long after the tube is removed.

Red rubber tubes can cause laryngeal necrosis and œdema (Iglauer and Molt, 1939; Olsen, 1948), and there is parallel evidence from urethral strictures occurring after the use of indwelling catheters.

The œsophagus at autopsy can show inflammation and ulceration in vertical strips along the projecting surface of the œsophageal folds. This is thin evidence that such inflammation follows the mechanical or chemical irritation of the tube, for few of these patients had symptoms of œsophagitis prior to death, and the changes can be as readily attributed to post-mortem digestion. Bartels (1935) examined 72 cases of œsophagitis seen at autopsy and found no difference in the appearance between those patients with indwelling tubes and those without.

5. *Debilitation.*—The distal œsophagitis seen in debilitated patients and at autopsy (Bartels, 1935; Vinson and Butt, 1936) can be severe, and can be associated with marked hæmatemesis (Pringle and Teacher, 1919).

The possibility that many instances of œsophageal erosion seen at autopsy resulted from post-mortem digestion has been discussed since John Hunter's paper (1835). This change is pathologically similar to gastric erosions rather than reflux œsophagitis, and there is little evidence that such a lesion would progress to stricture formation. However, in two of Douglas's (1956) patients, debilitation might have been a factor in the development of œsophageal stricture after intubation.

6. *Nicotinic Acid Deficiency.*—The œsophagitis seen in nicotinic acid deficiency (Fisher, 1944) could be significant here, for some degree of starvation accompanies duodenal stenosis; however, no other evidence of vitamin B deficiency was observed in these patients.

7. *Cerebral Damage.*—The hæmorrhagic ulceration of the œsophagus after brain-stem surgery, described by Cushing (1932) and Masten and Bunts (1934), appears to have no relevance to the present patients.

8. '*Post-operative*'.—Œsophageal stricture after abdominal operation has been described in a variety of contexts (Pringle and Teacher, 1919; Moutier, 1921; Bartels, 1935; Vinson and Butt, 1936; Vinson, 1941; Paul, 1943; Benedict, 1946; Bergquist, 1946; Mason and Ausband, 1952; Douglas, 1956).

Many factors have been present in many of these patients, particularly vomiting, intubation, and debilitation. This group forms such an ill-defined congeries that it is not possible to separate responsibilities except by opinion.

9. *Inflammatory.*—It has been suggested that thrombophlebitis from adjacent inflammatory foci such as cholecystitis or duodenal ulcer would extend to the intramural veins of the œsophagus and cause œsophagitis. This is unsupported by pathological evidence (Bartels, 1935). Similarly, Mosher (1931) and Moersch and Camp (1934) suggested that lymphatic spread of infection might be responsible for the inflammatory changes, but no other evidence has been adduced to confirm this.

These, then, are the possible mechanisms of œsophagitis with stricture in patients with an intact stomach. Regurgitation of acid pepsin might be a common effector mechanism in all instances, acting to greatest advantage on an œsophagus otherwise damaged.

In the 3 patients reported here, hiatus hernia, duodenal obstruction, vomiting, debilitation, and intubation could all have been important in inducing œsophagitis.

The single outstanding feature negating the responsibility of these factors is that stricture with dysphagia began for the first time *after* gastrectomy, and was then severe and progressive. Even had these factors been important, they would all be expected to have progressively less importance after gastrectomy.

Evidence of Alkaline Œsophagitis.—

1. The patients reported here developed œsophagitis with stricture *after* their gastric acid level had presumably been lowered by gastrectomy. Pepsin acts optimally at *p*H 1·5, and its action is virtually abolished at *p*H 5. Partial gastrectomy is often successfully used as a treatment for peptic reflux œsophagitis, the rationale being acid reduction and pepsin inactivation.

It is possible that some peptic œsophagitis preceded gastrectomy, but obstructive symptoms were absent before operation in all patients, and progressed with obvious severe œsophagitis after gastrectomy. These changes were so gross and dramatic and progressive that 'unmasking' of previous œsophagitis is untenable. The slow fibrosis of repair after peptic œsophagitis could induce late stricture formation, but ascending œsophagitis would be absent after gastrectomy, and the stricture would not be expected to progress proximally in a manner so typical of continuing œsophagitis.

This negative evidence of the lower gastric acidity in these patients after gastrectomy with resultant failure of peptic activity differentiates this group from those with an intact stomach. However, this lowering of gastric acidity is mainly presumptive evidence, for it is difficult to obtain reliable figures for acid levels after Polya gastrectomy. At the same time, this difficulty underlines the low probability of retention of a highly acid gastric juice.

2. Jejunal contents enter the stomach readily after gastrectomy. If gastro-œsophageal reflux occurs easily, as with coincident hiatus hernia, then bile and pancreatic juice can regurgitate into the œsophagus. Hiatus hernia was certainly present in 2 of these patients, and in the other easy reflux into the œsophagus was seen at œsophagoscopy. The 4 cases of œsophageal stricture described by Bingham (1958), occurring after gastro-enterostomy or gastrectomy, all had an associated hiatus hernia. McKeown (1958) described 2 cases with œsophageal stricture after uncomplicated gastrectomy, one of whom had hiatus hernia. The patient described by Benedict and Daland (1938) developed œsophageal stricture after partial gastrectomy complicated by gross efferent loop obstruction causing regurgitation of jejunal contents and requiring jejunojejunostomy for its correction.

3. Alkaline œsophagitis is seen after total gastrectomy with loop œsophagojejunostomy (Ripley, Olsen, and Kirklin, 1952; Kelly, Maclean, Perry, and Wangensteen, 1954; Pontes, Polak, and Campos, 1956; Helsingen, 1960a). Paulson (1953), using endoscopic methods, found that œsophagitis began early after total gastrectomy, and Nissen (1954) describes strictures occurring within a few months of total gastrectomy. This complication is uncommon if a long Roux loop is used in the œsophagojejunostomy (Allison, 1958). Jejunojejunostomy is not usually successful in relieving the alkaline œsophagitis after loop œsophagojejunostomy, for the jejunal contents continue to follow the path of normal peristalsis. Efskind (1955) combines a constricting tie around the afferent loop of jejunum between the œsophagojejunostomy and the jejunojejunostomy to force the flow of jejunal contents away from the œsophagus. This is often effective in preventing such regurgitant œsophagitis (Plenk, 1957; Helsingen, 1960b).

In an analogous fashion, McKeown (1958) was successful in his 2 patients in relieving the œsophagitis with stricture after gastrectomy by converting the Polya reconstruction to a Billroth-I type to divert the duodenal contents away from the distal œsophagus. That this single manœuvre was successful (for he bravely avoided repairing the hiatus hernia in the first patient) is supporting evidence of the mechanism of alkaline œsophagitis.

4. Cross and Wangensteen (1951) have induced experimental œsophagitis in cats and dogs by inundation of the œsophagus with bile and/or pancreatic juice, though Redo, Barnes, and de la Sierra (1959) found that bile alone did not produce œsophagitis. Hoag, Kiriluk, and Merendino (1954) regularly produced experimental alkaline œsophagitis in dogs and rats when gastrectomy, resulting in histamine-fast achlorhydria, was combined with abolition of the efficiency of the gastro-œsophageal sphincter. These experimental conditions parallel those after partial gastrectomy when hiatus hernia is also present.

In summary, there is good evidence that the severe progressive œsophagitis with stricture seen in these 3 patients after gastrectomy is similar in mechanism to the alkaline œsophagitis seen after œsophagojejunostomy.

How could this complication be avoided? In what way should routine gastrectomy be altered? First, it must be stated that the complication is rare. Only 7 other cases have been found in the literature, although McKeown implies that the complication occurs more frequently than is reported.

If the mechanism is truly one of gastro-œsophageal regurgitation, then an associated hiatus hernia should be repaired at the same time as gastrectomy, and efferent loop obstruction dealt with promptly if it occurs.

If peptic œsophagitis results from the combination of hiatus hernia and duodenal ulcer, then partial gastrectomy alone usually cures the œsophagitis; whereas repair of the hiatus hernia alone, in the presence of duodenal obstruction, is likely to fail. It is suggested, however, that partial gastrectomy be combined with repair of the hiatus hernia to limit the possibility of alkaline œsophagitis that could occur after partial gastrectomy alone. By the same token, even if no hiatus hernia has been demonstrated radiologically before operation, the hiatus should be examined at gastrectomy, and repaired if a sliding hernia can be demonstrated.

When the complication of œsophageal stricture has occurred, the problems are to deal with the stricture and with the regurgitation which might be through a sliding hiatus hernia or forced by outlet obstruction from the stomach. The stricture is difficult to control if the regurgitant cause continues. The jejunal contents can be separated from the œsophagus by repair of the hiatus hernia, or by conversion of the gastrojejunostomy to a gastro-duodenal anastomosis. Œsophageal dilatation might be necessary after such repair or reconstruction.

CONCLUSION

1. Œsophageal stricture is a rare but severe and progressive complication after partial gastrectomy for duodenal ulcer.

2. The œsophagitis and stricture are unlikely to be caused by acid-peptic regurgitation by vomiting, duodenal obstruction, or nasogastric tube.

3. The œsophagitis and stricture are probably caused by regurgitation of alkaline-tryptic jejunal contents into the œsophagus.

4. Sliding hiatus hernia or efferent loop obstruction can be mechanisms causing regurgitation of jejunal contents in these patients.

5. The treatment of such a stricture involves separation of the jejunal contents from the œsophagus by repair of the hiatus hernia, correction of efferent loop obstruction, or conversion of Polya to Billroth I gastrectomy. The stricture will require repeated dilatation.

SUMMARY

1. Three cases of œsophagitis with stricture occurring shortly after partial gastrectomy are presented.

2. The possible mechanisms of such œsophageal strictures are discussed.

3. It is suggested that regurgitation of alkaline jejunal contents provides the mechanism, and that treatment should be directed towards its cessation.

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ADENOID CYSTIC CARCINOMA

By J. KIRK, W. F. WALKER, and E. W. WALTON

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ADENOID cystic carcinoma is a term often used for a glandular carcinoma of low grade malignancy and characteristic histological features which occurs most commonly in the major salivary glands (Foote and Frazell, 1954). Similar neoplasms have been described, though less frequently, in other sites, in particular the minor salivary glands and the mucus-secreting glands of the oral cavity, paranasal sinuses, nasopharynx, trachea, and bronchi. The present case is of interest in its unusual mode of presentation and in that the mandible, larynx, and thyroid were extensively infiltrated by the neoplasm at the time of surgery; it is reported in order to support the view that adenoid cystic carcinoma is a separate entity, differing in behaviour from and with a graver prognosis than the much more common pleomorphic salivary adenoma with which it is often confused.

CASE REPORT

The patient, a female, was 47 years old when she first noticed a painful swelling under the right lower jaw in 1949. She was seen medically for the first time at another hospital in 1952 and a biopsy of the swelling was done. Normal muscle tissue only was obtained. Further investigations were refused.

She was seen again in 1957 when she had persistent pain in the lower jaw, redness and induration of the overlying skin, and trismus. A radiograph of the mandible showed a destructive lesion of the angle and body with pathological fracture (*Fig.* 337). The clinical picture suggested actinomycosis of the jaw and the patient was given penicillin with considerable symptomatic relief. Unfortunately, she did not report for follow-up and when next seen, in late 1958, the lesion had obviously progressed. She was admitted with bleeding from an ulcerating mass involving the body of the right mandible (*Fig.* 338). Pain and trismus were marked. Radiography of the mandible at this time showed extensive bony destruction with little reaction, suggesting a neoplastic lesion (*Fig.* 339). Biopsy revealed the presence of an adenoid cystic carcinoma (*Fig.* 340).

In January, 1959, excision of the lesion was attempted.

OPERATION.—The skin incision encircled the sinus area and extended backwards below the right ear and downwards along the anterior border of the sternomastoid

muscle to the sternal notch, and from there backwards along the medial two-thirds of the clavicle. Neoplastic tissue was found to extend through the mandible to involve the floor of the mouth; upwards to the base of the skull, soft palate, and lateral wall of nasopharynx; and downwards to the thyroid, and across the larynx with infiltration of the vocal cords. The right common carotid artery embedded in the tumour was thrombosed. A wide excision of the tumour was carried out involving a total laryngectomy. Neoplastic tissue left at the base of the skull was coagulated by diathermy. The lower posterior flap of skin was attached to the posterolateral wall of the pharynx. The skin on the left side of the neck was brought over and attached to the left side of the pharynx and sutured to the other flap of the skin below. A permanent tracheotomy was established. Split skin-grafts were placed on raw areas on the side of the cheek and above the clavicle (*Fig.* 341).

FIG. 337.—Comparison of both rami of mandible shows destructive process on right side.

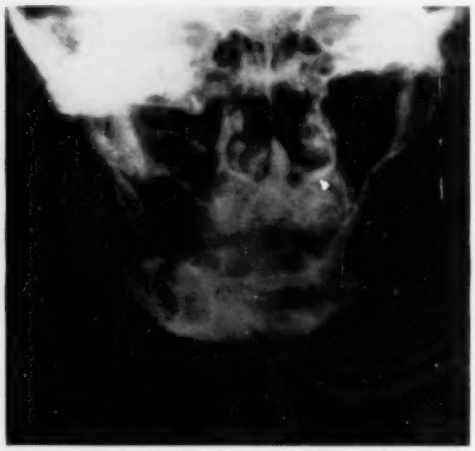

FIG. 339.—Extensive bony destruction right side of mandible prior to operation.

PATHOLOGY.—The whole of the surgical specimen was sent for pathological examination. It consisted of the following tissue:—

1. Three pieces of mandible: the head, a 4-cm. length of the ramus including the coronoid process and part of the condyloid process, and a 4-cm. length of the body, the medial border of which was frayed.

2. A complete larynx including the cricoid cartilage and hyoid bone. Overlying the superior horn of the right thyroid cartilage and the great horn of the hyoid bone was a mass of whitish neoplasm partly buried in muscle which extended downwards as a polypoidal growth over the right thyrohyoid membrane to involve both the vestibular and the vocal folds on the right side. The epiglottis, cricoid, and the remainder of the thyroid cartilage appeared normal.

3. A piece of tongue 3 cm. in diameter; the submucosa and muscularis were macroscopically infiltrated by whitish neoplasm.

FIG. 338.—Appearance of cheek prior to surgery in January, 1959.

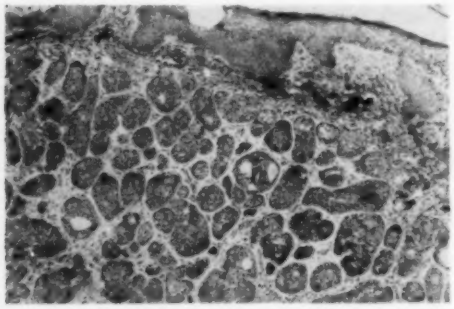

FIG. 340.—Biopsy of subcutaneous neoplastic tissue with the typical histological features of adenoid cystic carcinoma. H. and E. (× 28·5.)

4. A piece of thyroid tissue measuring 2 × 2 × 1 cm.

5. An irregular piece of soft tissue from the lateral pharyngeal wall and the base of the skull; it included part of the right styloid process, measured 5 × 2 × 2 cm., and was macroscopically widely infiltrated.

6. A ring of skin, on average 2 cm. wide and measuring 9 × 8 cm.; everywhere the subcutaneous tissue was infiltrated by neoplastic material.

7. Two irregular pieces of soft tissue each 1 cm. in diameter and each clearly infiltrated by neoplasm. Sections from each piece of tissue including the thyroid showed that all were infiltrated by a neoplasm with the characteristic histological features of adenoid cystic carcinoma (*Fig.* 342). The cribriform and cystic nature of the neoplasm was in places very striking. Though mitotic figures were few, there was infiltration of perineural lymphatics and also of blood-vessels, clumps of tumour cells being frequently found within the walls and lumina of small arteries and veins. Mucin secretion, as measured by the PAS, Best's mucicarmine, and alcian green methods, was very pronounced, with positively staining material filling the clefts and spaces between the anastomosing columns of tumour cells.

Reconstruction of the side of the neck was undertaken in spite of the doubt as to complete removal of the tumour, because of the discomfort of such a large oropharyngeal fistula.

METHOD OF REPAIR.—A marsupial type abdominal flap was used to close the defect, being transferred via the left forearm (*Fig.* 343). This provided both inner and outer cover for the fistula into the mouth and pharynx. A partial loss of the flap anteriorly below the chin was closed by means of an acromio-pectoral tube pedicle (*Fig.* 344).

The fistula was finally closed by September, 1959, but a biopsy in May, 1959, immediately prior to transfer of the abdominal flap, showed adenoid cystic carcinoma remaining in the floor of the mouth. Further extirpation

FIG. 341.—To show extent of oropharyngeal fistula after initial surgery.

of the tumour was not considered possible or an attempt desirable.

Radiotherapy was considered but the patient refused to have any further treatment.

POST-OPERATIVE COURSE.—She was discharged home in October, 1959—comfortable and free of pain. She had difficulty in taking solid diet because of her extreme trismus and some difficulty in swallowing. She managed liquids very well, however, and was given supplementary complan feeds.

Although signs of increasing tumour became obvious in the floor of her mouth, she continually refused radiotherapy. There was a slow deterioration in her health between January and April, 1960, with progressive difficulty in swallowing. She died in April, 1960.

AUTOPSY FINDINGS (Dr. W. W. Park).—The upper end of œsophagus was completely occluded by a mass of extremely dense neoplastic tissue. The base of the skull was completely clear of neoplasm. Metastases were present as several small (up to 1 cm. diameter) circular areas on surfaces of both lungs and as two nodules of 1·5-cm. diameter in the liver.

DISCUSSION

1. Incidence.—The incidence of adenoid cystic carcinoma is difficult to determine as this neoplasm is confused in many series with pleomorphic salivary adenoma on the one hand and various other glandular carcinomas on the other. However, McDonald and Havens (1948), in a study of 339 cases of glandular neoplasms occurring on the lips, nose, paranasal sinuses, and oropharynx, found that 86 of these were 'cylindromas'. Studies of neoplasms arising in salivary glands show that adenoid cystic carcinoma is one of the least common. Rawson, Howard, Royster, and Horn (1950) found 11 cases out of 160 neoplasms arising in all salivary glands. Quattlebaum, Dockerty, and Mayo (1946) found 21 cases out of a

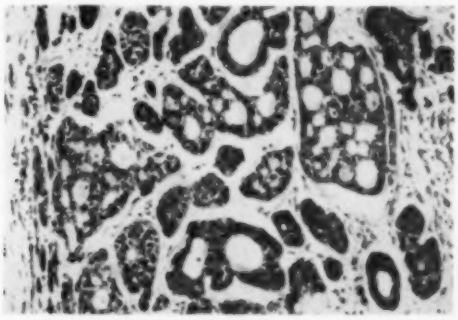

FIG. 342.—A higher-power view of the neoplasm to show the characteristic cystic and cribriform structure. H. and E. (× 62.)

FIG. 343.—To show lined abdominal flap prior to transfer to cheek defect.

series of 210 parotid neoplasms, while Vellios and Shafer (1959), in a study of 54 tumours of the intra-oral accessory salivary glands, found that only 8 were cylindromatous. On the other hand, in a careful study of ectopic salivary tumours summarized in a

FIG. 344.—After insertion of lined flap with closure of oropharyngeal fistula.

Hunterian lecture to the Royal College of Surgeons, Harrison (1954) found that of 82 cases seen at the Christie Hospital, Manchester, between 1937 and 1952, no less than 46 were 'cylindromas'. Of these 46 patients 16 were males and 30 females; 21 cases were in the palate, 4 in the floor of the mouth, 8 in the tongue, 2 in the buccal mucosa, 1 in the lower lip, 6 in the maxillary sinuses, 3 in the posterior nasal space, and 1 in the ethmoid sinus. In our case the exact site of origin could not be determined because of the late stage in the disease at which the patient came to surgery.

Adenoid cystic carcinoma arising from the minor salivary gland is, of course, less common than the same neoplasm in the parotid. Thus Wawro and McAdams (1954) found that of 18 cases, 12 occurred in the parotid, 2 in the submaxillary glands, 3 in the palate, and 1 at the base of the tongue. The site of origin did not affect the behaviour. Other authors have had the same experience.

2. Pathology.—The chief histological feature of adenoid cystic carcinoma is the arrangement of rather small darkly staining cells with relatively little cytoplasm, closely contiguous in anastomosing cords, between which are rounded acellular areas which may be empty or may contain mucus, hyaline, or mucohyaline material (Foote and Frazell, 1954). The constant resemblance to basal cells has led many in the past to call this neoplasm 'cylindromatous basalioma' or 'basal-cell carcinoma of salivary type'. Most authors are now agreed, however, that the neoplasm arises in mucus-secreting glands, though there is no clear evidence to incriminate either the cells of the ducts or acini.

3. Clinical Course.—Our case illustrates each of the typical features of adenoid cystic carcinoma: the slow rate of growth; the large size that the local lesion may achieve; the extreme invasiveness; the tendency to local recurrence, and the tardiness of distant metastasis. The delay in diagnosis in this case resulted from the too ready acceptance of the beneficial effect of penicillin as indicating an inflammatory lesion, and from the inadequate follow-up. Surgery when finally carried out, even though fairly extensive, was not in effect complete enough, with the result that recurrence at the upper end of the œsophagus led to death from inanition. The post-mortem findings indicated that more radical surgery might have increased the expectation of life, as the metastases were not sufficiently widespread to cause death for at least many months. It is unfortunate that radiotherapy was refused as 4 Mev therapy might have been of value.

4. Prognosis.—Adenoid cystic carcinoma is a far more serious disease than is generally realized in that local recurrence is usual and distant metastasis common. In their review of 86 cases McDonald and Havens (1948) found that at the time of review 22 patients had died from local recurrence, on average 7·4 years after the onset of treatment; 15 patients had developed distant metastasis, and in a further 8 the local lesion had recurred although the patient was still alive. Harrison (1954) found a local recurrence in 31 of his 46 cases, though the time interval had been as great as 12 years. Nine patients showed metastasis to the cervical lymph-nodes and 6 to distant organs—the lungs in 4, the liver in 1, the brain in another. In our case there was a local recurrence within 5 months of surgery and the patient died in April, 1960, 15 months after the operation and 11 years after onset of symptoms. In keeping with the common course of events in these cases there was extensive local recurrence and metastases were present in the lungs and liver.

SUMMARY

The present case in its location, prolonged clinical course, and histological features is a typical example of adenoid cystic carcinoma. This is the name which we believe describes most suitably the appearance and behaviour of this distinctive group of neoplasms. Furthermore, the somewhat archaic term 'cylindroma' which was formerly used is unsuitable, as it has been applied not only to neoplasms arising in salivary and mucus glands but also to various skin-appendage tumours which are of somewhat different structure and behaviour. This case strengthens the view that adenoid cystic carcinoma should be regarded, clinically and pathologically, as a malignant neoplasm, and not, as suggested by Willis (1956), as a variety of pleomorphic adenoma. Surgical excision of destructive tissue affords the best hope of cure.

Acknowledgements.—We are indebted to Professor D. M. Douglas for permission to publish this case, to Dr. W. W. Park for advice and autopsy report, to Mr. J. W. Corkhill who prepared the photomicrographs, and to Mr. M. Ettle for the clinical photography.

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STRANGULATION OF THE SMALL INTESTINE IN HOLES IN THE GREAT OMENTUM

A REPORT OF TWO CASES

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Herniation through the great omentum is rare, and when it occurs the omentum is usually already fixed to other viscera or to the abdominal wall, either by adhesions or by its inclusion in a hernial sac; herniation may also occur as a result of trauma. A review of the literature revealed only 20 recorded cases of spontaneous herniation of the intestine through a free great omentum. It was therefore deemed worth while to record a further 2 examples of this condition.

CASE REPORTS

Case 1.—W. A. K., a male aged 39 years.

On the morning of his admission, following breakfast, he complained of sudden severe colicky pain localized to the epigastrium. Two hours later the pain became more generalized, but less severe. The pain was accompanied by severe vomiting. The bowels acted in the morning, but this did not ease his pain and he did not pass flatus.

During the previous six months he had complained of vague indigestion, consisting of an epigastric aching pain which might last the whole day.

His past history was unrevealing and in particular he had not undergone any previous abdominal operation.

On Examination.—He was shivering and sweating and appeared ill. Temperature normal, pulse 80/min., blood-pressure 150/90. The abdomen was not distended and visible peristalsis was not present. There was generalized guarding, but local tenderness was absent and abnormal masses could not be palpated. Bowel-sounds were absent. Herniæ were not detected. Rectal examination revealed tenderness in the rectovesical pouch.

At Operation.—The abdomen was opened through an upper abdominal midline incision, and on entering the peritoneal cavity a moderate quantity of serosanguineous fluid was found. Further exploration revealed that a loop of jejunum 10 in. (25 cm.) in length had passed forwards through a hole in the great omentum near its free border. This loop was markedly thickened and dark red in colour. The defect in the great omentum was 2 in. (5 cm.) in diameter, and its lower border was formed by a slender strand ¼ in. (6 mm.) thick. No adhesions or internal herniæ were found.

The band causing the obstruction was divided and the abdomen was closed. The patient made an uneventful recovery and was discharged on the eleventh day.

Case 2.—G. H. L., a male aged 50 years.

During the evening prior to admission he experienced sudden colicky pain in the centre of his abdomen, which persisted and was associated with vomiting. The bowels acted in the morning, but he did not pass flatus afterwards. There was no past history of similar pain or of any abdominal operation.

On Examination.—He was writhing with pain and was vomiting. Temperature 97° F., pulse 64/min., blood-pressure 140/85. The tongue was coated and the breath foul. Neither abdominal distension nor visible peristalsis was present, but palpation disclosed generalized guarding and tenderness; no abnormal masses or herniæ were detected. Bowel-sounds were increased.

At Operation.—Serous fluid was present in the peritoneal cavity. There was a short congested segment of intestine in the lower part of the jejunum, with petechial hæmorrhages under the serosa, sharply limited above and below by dark oblique bands. In the free edge of the great omentum, at the junction of middle and left third, there was a defect 1¼ in. (3 cm.) in diameter, its free margin consisting of a strong fibrous cord 2 mm. in diameter; the rest of the margin was rounded, firm, and hyperæmic; a fibrinous deposit on its surface suggested that it had been responsible for the obstruction. The segment of gut fitted tightly into this aperture, the oblique bands corresponding to the constricting margin. The findings were consistent with strangulation and spontaneous reduction of the affected loop of intestine. Neither adhesions nor internal or external herniæ were present.

Excision of the relevant portion of the great omentum was carried out and the abdomen was closed.

The patient made an uneventful recovery and was discharged on the tenth day.

DISCUSSION

In the *Medical Times* of London of Saturday, July 18, 1840, there appeared the following report by 'A. B.':—

"Jane Rushbrook, aged 49, had been confined to bed for three years, with cancer of the uterus. Previously to her death the poor patient complained of violent heat, and pain in her bowels; she vomitted, at intervals, a fæcal smelling fluid; the bowels could neither be moved by the strongest purgatives, nor by injections; the pulse was quick, wiry and easily extinguishable; the tongue red, dry, and brown down the centre; she complained of intense thirst, but the stomach rejected everything, fluid or solid."

After two days she died and at post-mortem examination "It was found that one of the small intestines had protruded itself through the great omentum, and become strangulated by it."

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This is the earliest record of this type of obstruction.

Sir Frederick Treves (1899), in his book on *Intestinal Obstruction*, refers to two specimens of this condition, and one of these may be still seen in the Pathology Museum of University College Hospital Medical School, and is identified easily from the original drawing (*Fig.* 345).

The literature on this cause of obstruction has been reviewed fully by Martzloff (1930) and Pampari (1948). Martzloff collected 15 recorded cases and reported his own case. Pampari collected a further 18 recorded cases and added 1 personal case. He analysed all recorded cases and found that in 8 a part of the omentum had been caught in a hernial sac; in 3 there was a history of trauma and in 3 a history of previous operation; 4 cases were associated with previous inflammatory disease; the remaining 17 cases he classified as 'spontaneous or congenital'.

FIG. 345.—Omental strangulation of small intestine as illustrated by Sir Frederick Treves.

Another case was recorded by Sanes and Postoloff (1944), and Lunzenauer (1954) recorded 2 cases admitted in Leipzig within one week. These cases were all 'spontaneous'.

The small intestine was affected in all cases except that of Gorski (1898), where the large intestine was involved, and that of Lasichak (1950), where part of the free border of the omentum itself had become strangulated in a hole in the omentum. In all cases the herniation has been single with the exception of the case recorded by Le Fort (1879), of a young man who developed intestinal obstruction following a kick from a horse. At post-mortem examination the man was found to have two herniæ of portions of small intestine through separate slits in the omentum.

The aetiology of spontaneous herniation of the intestine through an aperture in the great omentum is debatable. When there is no history of previous trauma, inflammation, or operation, and the omentum is found to lie free within the peritoneal cavity, the possible causes of the condition are congenital defect in the omentum or rupture due to trauma too trivial or too distant in time to have been remembered by the patient. The theory of congenital defect is the most probable and was first advanced by Treves (1899). The great omentum is developed from the dorsal mesentery of the stomach; as the stomach rotates during fœtal life its mesentery enlarges first to the left and then over the transverse colon, and the anterior and posterior walls of this caudal part of the lesser sac become fused together to form the great omentum. In favour of the congenital theory is the fact that herniation through holes in the mesentery of the small intestine also occurs and is equally rare; the literature on this condition has been reviewed fully by Blandy (1960), and most authors consider it to be due to congenital defect. Against the congenital theory is the fact that as the great omentum, unlike the mesentery of the small intestine, is formed by the fusion of two layers of mesentery, it pre-supposes that two separate congenital defects must overlie each other. In view of the rarity of the condition, this supposition is acceptable.

SUMMARY

Two cases of spontaneous strangulation of small intestine in holes in the great omentum are presented, together with a brief review of the literature.

I wish to thank Mr. N. M. Matheson for permission to publish these cases, and Cassell & Co. Ltd. for permission to reproduce the drawing from Sir Frederick Treves' book.

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RHABDOMYOSARCOMA OF THE EPIDIDYMIS

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THE outstanding feature of rhabdomyosarcoma is its paradoxical occurrence in situations where striated muscle is rare. The tumour has been described most commonly in the urogenital tract (vagina, urethra, bladder, prostate, spermatic cord, and epididymis) and in the upper respiratory passages, and there have been occasional reports of its occurrence in lungs and bile-ducts. The spermatic cord and epididymis do normally contain small amounts of striated muscle, and in other sites, for example, the palate, there is closely related voluntary muscle, but these are minute in relation to the bulk of the skeletal musculature. In view of the rarity of malignant tumours in skeletal muscle the suggestion that rhabdomyosarcomata arise from the primordium of local muscle, or from displaced myotome tissue as suggested by Wilms (1900), is unconvincing. Their bizarre and widespread distribution is more compatible with an origin in embryonic mesenchymal tissue which has undergone aberrant differentiation (Willis, 1948).

A common histological feature which would support such an origin is the presence of myxomatous tissue, resembling primitive mesenchyme. This is well seen in the present example, in which the tumour is for the most part poorly differentiated and anaplastic; indeed, the diagnosis here rests on the finding of a few striated muscle-fibres. One may therefore infer that in other more anaplastic examples of this neoplasm it may be impossible to make a more than tentative pathological diagnosis. It is of interest that highly anaplastic tumours whose origin is doubtful may have their muscular nature revealed in their metastases (Hirsch, 1934). Such secondary deposits probably result from the dissemination of primitive anaplastic tissue which then differentiates to form muscle cells (Cappell and Montgomery, 1937).

The rarity of this condition makes it difficult to estimate the prognosis in a particular case, and study of the relevant literature shows that, apart from examples of rapidly fatal tumours, most case reports have been published shortly after operation, and these fail to allow adequate time for follow-up and assessment of prognosis. Prognosis therefore tends to be based rather pessimistically on the behaviour of the most malignant forms. A rather more optimistic view is suggested by the present report of a patient who is still alive and well seven years after removal of the primary tumour.

CASE REPORT

A male, aged 16 years, was first seen in 1954. He complained of a swelling in the left side of the scrotum, present for 7 days. On the day before he first complained of the swelling he had received a blow on the scrotum from the starting handle of a car. Closer questioning, however, revealed that there had been a swelling present for a few weeks before the accident.

ON EXAMINATION.—An 8 × 6-cm. elliptical mass was felt in the left side of the scrotum (*Fig.* 346). It was hard, not tender, had an irregular surface, and at the upper pole

FIG. 346.—Tumour on left side of scrotum.

anteriorly there was a smaller protuberance about 3 × 3 cm. which was soft and tender. The whole mass was mobile within the scrotum. There was no associated enlargement of lymph-nodes. The pre-operative diagnosis was merely 'tumour of the testis'. The Aschheim-Zondek test was negative.

OPERATION.—The mass was exposed through a scrotal incision passing into the left iliac fossa. The tumour extended along the spermatic cord to the superficial inguinal ring. Orchidectomy was carried out with removal of the cord as far as the deep inguinal ring, where it was felt to be normal.

PATHOLOGICAL DESCRIPTION.—

Gross Specimen (*Figs.* 347, 348).—The specimen consists of a rounded creamy-white tumour 7 × 7 × 5 cm., the testis, and a short length of spermatic cord. The testis, which lies at the upper pole of the tumour mass, is compressed, but tumour and testis are separated by a well-developed fibrous capsule which invests the tumour. The capsule is deficient at the upper pole, where a small mass of tumour grows along the spermatic cord for about 1 cm. On its outer aspect the tumour is somewhat bosselated, and on section presents a whorled pattern.

Histology.—The tumour is of variable cellularity, consisting in part of closely packed masses of anaplastic cells, and in part of broad bands of poorly cellular collagenous fibrous tissue.

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In the most cellular zones (*Fig.* 349) the tumour cells are polygonal, have scanty cytoplasm, and rounded or elongated nuclei. Most nuclei have finely stippled chromatin, some are deeply pyknotic. In the less cellular areas, sections stained with phosphotungstic-acid hæmatoxylin (*Fig.* 351). In many muscle cells, particularly the plump round cells, there is marked vacuolation. That these vacuoles contain glycogen is shown by positive reactions

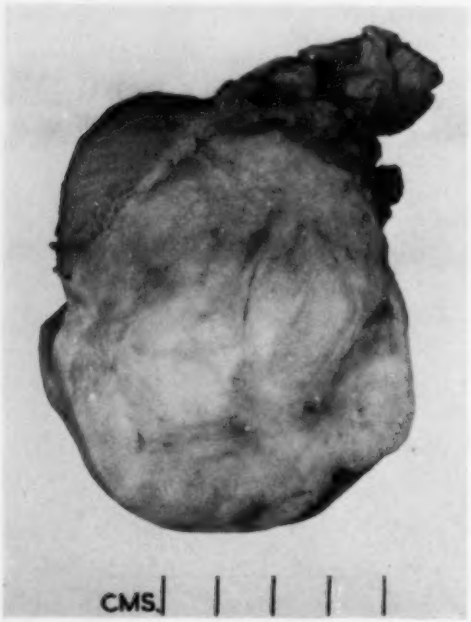

FIG. 347.—Tumour bisected showing testis lying at upper anterior pole.

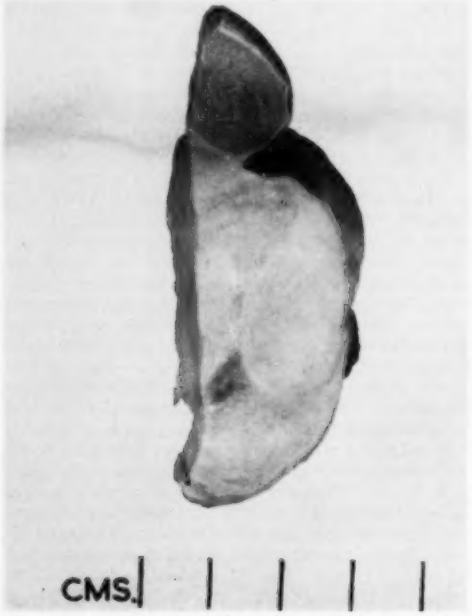

FIG. 348.—Dissected specimen, illustrating clear margin between tumour and testis.

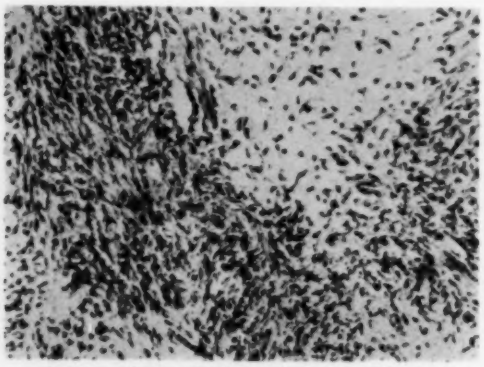

FIG. 349.—Section showing, right, poorly cellular myxomatous tissue and, left, highly cellular anaplastic tumour. H. and E. (× 175.)

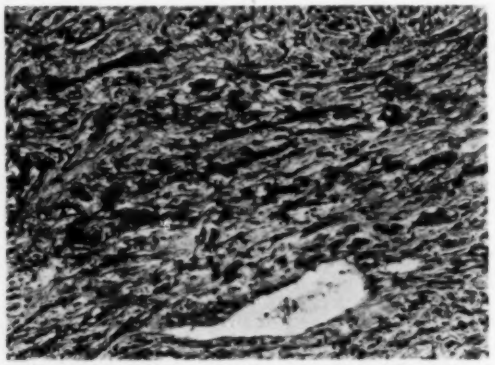

FIG. 350.—Portion of tumour showing many 'strap' forms. In few of these was striation demonstrable. H. and E. (× 100.)

the neoplastic tissue is of two main types: (1) loose myxomatous tissue with few evenly scattered cells, which have scanty cytoplasm and elongated or stellate nuclei; and (2) fibrous tissue in which there are numerous tumour cells. Some of these are obvious muscle cells and occur in small groups (*Figs.* 350, 351). A few strap-like forms are seen; many are shorter, oval, or rounded. Their cytoplasm is abundant and is most often quite smooth and hyaline. It sometimes exhibits longitudinal striations, but only rarely does one find cross-striations, even in with Best's mucicarmine and the periodic acid Schiff method, both of which are negative after incubation with diastase. The cytoplasm also stains positively with lissamine tartrazine (Lendrum, 1951). Many of these cells are multi-nucleate, with the nuclei lying peripherally.

There is a deep invasion of the tumour capsule, and in parts actual penetration. The tubules of the epididymis are embedded in tumour, but appear intact and contain spermatozoa along with other cells and debris.

The tumour has many well-formed arteries, veins, and lymphatics. Many of the lymphatics and some of the veins contain dense masses of highly anaplastic tumour tissue (*Fig.* 352).

COMMENT

Tumours arising in the scrotum, which are found to contain striated muscle, may be either primary muscle tumours or teratomata. Many examples reported as rhabdomyosarcoma of the cord and epididymis have involved the testis, and in these it is not possible to exclude a teratomatous origin. In only 6 of the 14 cases reviewed by Hirsch in 1934 were the growths separate from the testis. Spermatic cord tumours (Cappell and Montgomery, 1937; Shivers, 1944; Hansen, Enos, Park, and Postell, 1952) are more common than tumours of the epididymis (Strong, 1942), but they appear to be identical morphologically and may be considered together. However, by virtue of their site, tumours of the epididymis are more prone to invade the testis and thus give rise to difficulty in distinguishing them from teratomata. In the present case separation was clear-cut, and, since many blocks from all parts of the tumour were examined, all showing the same appearances, one can exclude a teratomatous origin with some confidence.

One of the prominent histological features in this tumour is the invasion of lymphatics and, to a lesser degree, of veins. The invading tissue is of the most primitive and anaplastic type, and one might reasonably expect early and widespread metastases. This is certainly the experience with rhabdomyosarcoma in general (Cappell and Montgomery), but as already mentioned, it is unwise to assess the overall prognosis of the group by its most malignant examples. It is probable that, as with other types of malignant neoplasm, there is a wide range of behaviour. Thus, further inquiry into the fate of the cases reported by Strong (1942) and Cannon, Altheide, and Allen (1956), one and two years respectively after excision of their tumours, reveals that both patients are now known to have been alive and well six years later (Strong, 1960; Cannon, 1960). Cannon reports that his patient, now 9 years of age, is still well and shows no evidence of metastases.

In the present case, surgical removal was not extensive, and did not include dissection of lymph-nodes. It was followed by post-operative irradiation, despite the fact that rhabdomyosarcoma is thought to be relatively radio-resistant. The histological features led us to expect dissemination of the growth and a rapidly fatal course—especially since the neoplasm was seen to invade the veins. In the event, the boy has survived seven years and remains in good health. This must make us suspect the validity of histological evidence of venous invasion as indicating an invariably fatal dissemination of a neoplasm throughout the body.

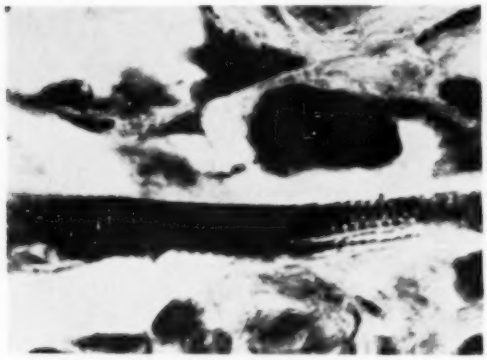

FIG. 351.—Strap-shaped cell, showing cross-striation. Phosphotungstic-acid hæmatoxylin. (× 700.)

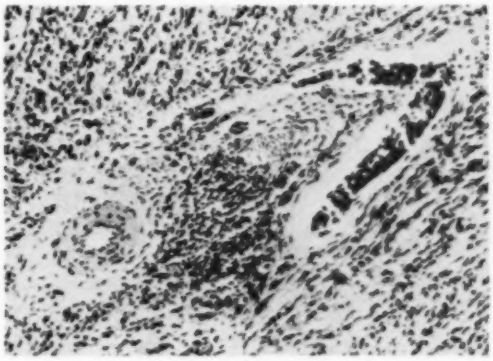

FIG. 352.—Undifferentiated neoplastic tissue in a vein. H. and E. (× 175.)

SUMMARY

A case of rhabdomyosarcoma of the epididymis is described. The patient is still alive and well, seven years after operation. It is suggested that the literature gives an unduly pessimistic view of these tumours.

We wish to thank Mr. W. G. Campbell for permission to publish this case.

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PILOID GLIOMAS OF THE ANTERIOR OPTIC PATHWAYS

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More than 400 cases of 'optic nerve gliomas' involving the nerve or nerve and chiasm have been reported in the literature. There might be little point in recording more if the clinical picture had been made clear or if a generally accepted form of treatment had emerged. They are rare tumours and it is unlikely that any one surgeon will see many cases in a working lifetime. Dodge, Love, Craig, Dockerty, Kearns, Holman, and Hayles (1958) reported the largest series from one unit when they collected 46 cases from the Mayo Clinic records of the last 40 years.

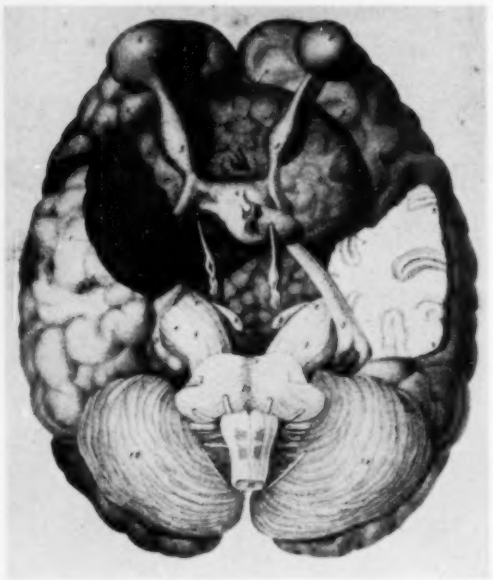


Fig. 353.—**1**, Outline of the tumour; **4**, Irregular mass of chiasm and tuber cinereum with pituitary stalk; **5**, Oval tumours of both optic nerves; **6**, Both globes with the right side enlarged and containing tumour. (*Figs.* 353, 354 *reproduced from 'Fungo midollare dell'occhio,' by B. Panizza.* 1821.)

This paper presents 10 cases with histological verification in 9. In these 9 cases the histology was virtually identical but the tumours did not all behave in the same way. By surveying these cases and the relevant literature it was hoped to understand more fully the effects of the age of onset and the site of the tumour on its natural history; whether growth was episodic or whether the tissue responses to the tumour varied; what effects total or subtotal removal or radiation had on the tumours with an apparently long benign course, and so whether any treatment was indicated (*see Table I*).

A new fronto-orbital osteoplastic flap is described for exposing and removing the whole length of the optic nerve.

THE LITERATURE

In the early literature all stages of the disease process are portrayed for surgery rarely interfered with the natural history. The best pathological demonstration was probably the first ever recorded—that of Panizza (1821), who presented descriptions and drawings of the brain of a 6-year-old girl (*Figs.* 353, 354). A huge tumour of the optic chiasm obliterated the third ventricle and the foramen of Monro to produce internal hydrocephalus, and extended along the thickened optic nerves to invade the proptosed eyeball on one side.

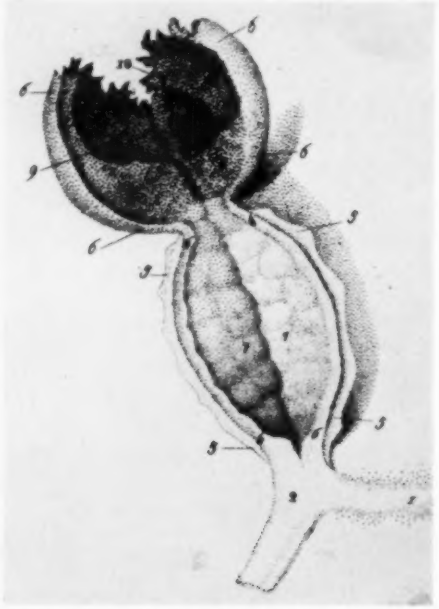


Fig. 354.—**1**, Chiasm; **2**, Enlarged right optic nerve; **5**, Envelopment of dura mater; **6**, Sclera continuous with inner sheath of optic nerve; **7**, Reddish, granular degeneration with no trace of nerve-fibres; **8**, Soft white tumour occupying the posterior half of the globe.

Blindness and proptosis were the only recognizable manifestations of the disease 150 years ago. The ophthalmic surgeons were therefore the first to see the cases, and they recorded them in the ophthalmic literature of the nineteenth century. These oculists knew that the tumour spread intracranially along the nerves, but they also knew that they could save a life by removing a proptosed and infected eye and prevent fatal panophthalmitis. Some died postoperatively and many of those coming to postmortem had intracranial extensions. Although the removal of the tumour was often known to be incomplete when eyes were removed and tumorous

nerve cut through at the apex of the orbit, some patients survived this and lived for another twenty years with the tumour apparently causing no more trouble (cases quoted by Hudson, 1912).

The first successful operation for the removal of an optic nerve tumour was performed in Edinburgh by Wishart (1833), and Knapp (1879) first performed a retrobulbar excision with preservation of the globe. With the advent of neurosurgery many years later attention was no longer confined to the orbit, and from tissue culture. Elvidge, Penfield, and Cone (1935) coined the term 'piloid astrocytomas', but Lundberg (1936) described 9 cases examined histologically as oligodendrogliomas. Del Rio Hortega (1940) concurred with the earlier designation and stated that the tumours belonged to a *group* of piloid astrocytomas occurring in the cerebellum, the medulla, and the corpus callosum.

The association of gliomas of the anterior optic pathways with neurofibromatosis has given rise to

Table I.—FOLLOW-UP OF 10 CASES OF PILOID GLIOMA OF THE ANTERIOR OPTIC PATHWAYS

CASE	AGE OF ONSET OF SIGNS	HISTOLOGICAL VERIFICATION	SITE OF TUMOUR. C, CHIASM RON, RIGHT OPTIC NERVE LON, LEFT	PRESENCE OF CAFÉ-AU-LAIT PATCHES SUGGESTING VON RECKLINGHAUSEN'S DISEASE	SURGICAL REMOVAL	FOLLOW-UP YEARS SINCE TIME OF ONSET	FINAL RESULT	RESIDUAL VISION L	RESIDUAL VISION R
1. M. S. (F) 45 222	1½ yr.	+	C + RON	+	Incomplete	17	Alive	J.1	Nil
2. J. R. (F) 297679	2 yr.	+	C + Both ON's	+	Nil	15	Alive	J.20 (Vision improved spontaneously)	J.10 (Vision improved spontaneously)
3. M. B. (M) 46 394	38 yr.	+	C + Both ON's	—	Biopsy only	18	Alive	J.6 (Vision improved spontaneously)	J.18 (Vision improved spontaneously)
4. J. S. (F) 59 12479	2 yr.	+	C + Both ON's	+	Incomplete	8	Alive	Nil	J.1
5. J. F. (F) 290361	27 yr.	+	LON	—	Complete (Globe left)	7	Alive	Nil	J.1
6. L. E. (F) 54 5958	1½ yr.	+	C + Both ON's	+	Incomplete	9	Alive	J.2	Nil
7. D. B. (M) 54 30683	1½ yr.	+	LON	+	Incomplete (Recurrence)	6	Alive	Nil	J.1
8. P. McG. (F) 54 33113	5 yr.	+	LON	—	Complete	7	Alive	Nil	J.1
9. J. A. (F) 38 208	7 yr.	+	C + Both ON's	+	Nil	6	Dead	? Nil (Deterioration in terminal stages)	? Nil
10. K. S. (M) 112455	19 yr.	+	C	—	Biopsy only	13	Dead	Nil	J.4

Martin and Cushing (1923) operated upon gliomas of the intracranial portion of the optic nerve and chiasm.

The clinical picture was partly formulated by Byers (1901) from the 81 case reports he was able to collect from the literature but he made no attempt to distinguish the various histological types of the primary tumours of the optic nerve. Virchow (1866) had established that at least some of these tumours were of glial origin, but other authors of the nineteenth century, impressed by the size to which these tumours grew and the cystic degeneration seen on cross-section, had variously called them 'myxomas', 'myxosarcomas', 'fibrosarcomas', or 'angiosarcomas', and those optic nerve tumours occurring together with von Recklinghausen's disease were regarded as neurofibromas, analogous with the swellings on the peripheral and other cranial nerves. The most important milestone in the understanding of the pathology came when Hudson (1912) reviewed 154 cases in the literature and in retrospect was able to classify them into two types, the meningiomas (28 cases) and the gliomas (118 cases).

Hudson's classification was later accepted and with the development of specific staining techniques the tumour cells were more accurately defined. Verhoeff (1932) and Russell and Bland (1934) described the tumours as 'astrocytomas', the latter authors reinforcing their conclusions with evidence

some controversy since it was first described by Michel (1873) and thought to be coincidental. Seventy years later Levitt (1940) wrote that ". . . optic nerve gliomas occurring without any other abnormality will be considered the only manifestation of neurofibromatosis". But the association of this tumour with any other manifestation of neurofibromatosis is not invariable. Von Recklinghausen (1882) who fully described neurofibromatosis and whose name was given to the condition by the French—although Smith (1849) of Dublin deserves the honour—personally examined 2 cases of optic nerve gliomas operated upon by Langenbeck and made no mention of neurofibromatosis (von Graefe, 1864).

THE CLINICAL PICTURE

The gliomas of the anterior optic pathways are mostly tumours of childhood and about seventy-five per cent present in the first decade and ninety per cent in the first two decades. The oldest case recorded presented at the age of 60 years. The tumours appear to arise anywhere from the chiasm to the optic disk and in no place particularly, and some are reported to have been multiple (Hessdorfer, 1883; Willemer, 1879).

Failing vision in one eye is usually the first symptom if the patient is old enough to complain, and the loss of vision may come so early in life that binocular

vision does not develop and a non-paralytic strabismus results. The degree of visual loss varies but the majority have no perception of light in the affected eye when first seen (Byers, 1901). In some cases useful vision persists for many years in the affected eye in spite of clinical progression of the tumour in other respects and bilateral total blindness is unusual even in the late stages of the disease. In 2 early cases recorded by Dodge and others (1958) the acuity had remained normal in both eyes when first seen. The pupil fails to react directly to light if the eye is blind.

The development of axial proptosis, which is non-pulsatile and irreducible, may follow next, but the delay in its appearance will vary with the site of the tumour and with its rate of growth. The proptosis may first bring attention to the unilateral loss of vision but the atrophic disk seen ophthalmoscopically will suggest that the loss is of long standing and hitherto unnoticed. Many records of the nineteenth-century cases coming late to surgery tell of gross proptosis with the globe forced out of the orbit by the tumour and the eyelids closing behind it (Aub, 1884).

Loss of external ocular movements and the presence of pain are unusual features but will depend upon the degree of proptosis. No case has been recorded where a glioma of the nerve has invaded or enveloped the ocular muscles or nerves to produce ocular palsies as happens with the optic nerve sheath meningiomas in the orbit. Any limitation of movement that does occur is presumed to be mechanical and due to the bulk of the tumour. Pain is difficult to attribute to any specific process.

Asymmetry of the face may be a concomitant of associated neurofibromatosis where an agenesis of one sphenoidal wing or a congenital cyst of the middle cranial fossa deforms the temple on one side. One orbit may increase in size to accommodate the tumour within it; the best examples occurring in the early nineteenth century when tumours were commonly compared in size with goose eggs and one reached a diameter of 7 cm. before removal (case quoted by Hudson, 1912). Plexiform neurofibromatosis of the branches of the oculomotor nerve may enlarge the orbit and in addition give rise to buphthalmos and irregular colobomata. Davies (1939) described this together with an extensive glioma of the whole optic nerve and chiasm.

One or both optic disks are almost always abnormal ophthalmoscopically and the commonest finding is optic atrophy, either primary or secondary. Papillœdema, either alone or in combination with optic atrophy, may occur in one eye or both. The presence of a large intra-orbital tumour with proptosis may cause venous obstruction and papillœdema on one side. Raised intracranial pressure due to ventricular obstruction by a chiasmal tumour may produce bilateral papillœdema and, if the obstruction is relieved, leave secondary atrophy superimposed on the primary atrophy produced by the compression of the optic nerve axons by the tumour cells. Fowler and Matson (1957) described 2 cases with optic atrophy in one eye and papillœdema in the other, mimicking the 'Foster-Kennedy syndrome'.

The tumour may be seen at the optic disk with the ophthalmoscope. Saebø (1949) described a white mushroom-like swelling of the disk later found to be due to glial cells in continuity with a glioma of the nerve. Fowler and Matson (1957) described disk involvement by tumour. Verhoeff (1932) presented a cut section of globe and nerve with involvement of the retina by tumour. Boles, Naugle, and Sampson (1958) showed a fundus painting of the disk with retinal involvement. McLean (1937) showed a picture of a histologically verified glioma of the retina without any tumour in the optic nerve or disk. Whether this was the retinal analogue of the glioma of nerve is difficult to say but it appears more related to the histologically similar phakomas and sub-ependymal nodules of tuberous sclerosis.

No typical deformity of the visual field occurs but concentric constriction usually accompanies the diminished visual acuity. Bizarre fields may occur early; altitudinal defects (Spaulding, 1958) and clear-cut bitemporal hemianopia (Martin and Cushing, 1923) have also been described.

Disturbance of the hypothalamic and pituitary function with large tumours of the chiasm gives rise to many symptoms and signs. Hypersomnia, polydipsia, and polyuria (Martin and Cushing, 1923; Cushing, 1930), and precocious puberty (Fowler and Matson, 1957; Dodge and others, 1958) are extreme forms, but obesity and some degree of eunuchoidism are common. The fully developed adiposogenital dystrophy and the panhypopituitarism of a Rathké pouch tumour have not been described.

The development of *athetosis* in 1 case described by Jefferson (1940) was attributed to diencephalic invasion from the chiasm. This patient also had epilepsy.

Raised intracranial pressure due to ventricular obstruction may be present when the patient is first seen. In the fatal cases of the past this was the common mode of death.

Evidence of von Recklinghausen's disease may exist. Crowe and Schull (1953), from a random survey of over 7000 adults, have suggested that the minimum cutaneous manifestations found in association with other manifestations of von Recklinghausen's disease are six café-au-lait patches of more than 1·5 cm. in diameter. In early childhood neither patches nor subcutaneous fibromata may have developed fully, but supporting evidence, if the condition is suspected in a child, may come from a sibling or parent with stigmata better developed. If a child has optic atrophy, especially unilateral, and neurofibromatosis there is most likely a glioma present on the anterior optic pathways. The combination of neurofibromatosis and glioma of the chiasm occurring in 2 siblings has been reported twice (Fowler and Matson, 1957; Davies, 1940).

Good prognosis with very slow growth of the tumour and long survival of the patient is common. The tumour may reveal itself in the 1st and still be little more obtrusive in the 20th year of the patient's life. As yet, no case has been recorded with a longer follow-up than 24 years.

RADIOLOGICAL FEATURES

The optic canals and the chiasmal sulcus of the sphenoid bone may develop abnormally in the presence of an enlarged optic nerve or chiasm. The axis of the optic canal changes in childhood up to

the age of 17 years (Goalwin, 1927) and allowing for this it may be possible to detect smooth, even enlargement of one or both canals radiologically. There is considerable individual variation in the size of the canal on each side, and a difference in diameter of one-third between the two sides may be found in the normal subject (Fowler and Matson, 1957).

Seen laterally, a deepening of the chiasmal sulcus and, with the enlarged optic canals, a narrowing of the bases of the anterior clinoid processes, gives a bilocular appearance to the floor of the sella, which then appears like the cross-section of a pear-gourd (Cushing, 1930).

Neither the enlarged optic canals alone nor the sella changes alone are specific for this condition (Timme, 1921; Olivercrona, 1932; Pfeiffer, 1943; Holman, 1959) and an optic canal of normal size radiologically does not exclude the extension of tumour through the canal. A 'gourd' sella together with enlargement of one or both canals should mean a glioma of the chiasm and nerves. Calcification in a tumour of the chiasm has been reported but is rare (Cushing, 1930; Bailey and Herrman, 1938; Lindgreen and Dichiro, 1951).

In addition to these changes seen on the plain radiographs there may be the radiological signs of raised intracranial pressure if the ventricular system is blocked. The 'beaten-silver' appearance of the increased convolutional markings on the skull vault, erosion of the dorsum sella, and sprung cranial sutures are characteristic.

Air encephalography and air or myodil ventriculography have demonstrated filling defects in the anterior wall of the third ventricle and the chiasmal cistern. Orbital (carotid) angiography may show displacement of the ophthalmic artery in the orbit (Grino and Billet, 1949) and orbital pneumatography may outline the intra-orbital tumour (Dubilier, von Gal, Freemond, and Evans, 1956).

SUMMARY OF CASES

Case 1.—Born with von Recklinghausen's disease: glioma of chiasm and right nerve at 1½ years: incomplete removal and no radiation therapy. Remains well at 18 years with normal vision in the remaining eye.

Case 2.—Born with von Recklinghausen's disease: glioma of chiasm suspected at 2 years and seen at operation at 10 years. (No biopsy possible.) No part of tumour removed and no radiation therapy given but remains well at 17 years with spontaneous *improvement* of vision.

Case 3.—No evidence of von Recklinghausen's disease: glioma of chiasm and right nerve presenting at 38 years and confirmed by biopsy at 42 years when almost blind. No radiation therapy given but remains well with spontaneous improvement of vision at 56 years.

Case 4.—Born with von Recklinghausen's disease: glioma of chiasm and left nerve probably from birth and confirmed by biopsy at 7 years. Incomplete removal and no radiation therapy but remains well with normal vision in the right eye at 10 years.

Case 5.—No evidence of von Recklinghausen's disease: glioma of left optic nerve confined to the orbit at 27 years. Total removal of optic nerve by combined orbitofrontal exposure with preservation of the globe. No radiation therapy given. Remains well with no evidence of recurrence at 34 years.

Case 6.—Born with von Recklinghausen's disease: glioma of chiasm and both nerves with proptosis and blindness on right at 18 months. Developed raised intracranial pressure and papillœdema at 2 years that was not due to ventricular obstruction. Swollen chiasm explored at 2 years and right eye removed at 3 years. No radiation therapy was given. Remains well at 10 years with no clinical evidence of advancement and good vision in remaining eye.

Case 7.—Born with von Recklinghausen's disease: extensive glioma of left nerve at 2 years removed together with the globe in two stages but some tumour left behind in the chiasm. At 7 years partial removal of a recurrence of the tumour in the nasal sinus presenting subcutaneously at the nasion. No radiation therapy given. At 9 years no further evidence of continued growth and normal vision in remaining eye.

Case 8.—Born with von Recklinghausen's disease: glioma of the left optic nerve with proptosis at 5 years. The whole length of the optic nerve was exposed by *the orbitofrontal bone flap* (*see Figs.* 357–360) and the whole nerve and globe removed in continuity from the chiasm. No radiation therapy was given. No evidence of recurrence after 7 years.

Case 9.—Born with von Recklinghausen's disease: glioma of chiasm at 7 years producing optic atrophy and precocious puberty. No operative or radiation therapy was given. Died at 13 years from internal hydrocephalus and brain sent from elsewhere for examination.

Case 10.—No evidence of von Recklinghausen's disease: glioma of chiasm at 19 years giving field defects, at 27 years giving hypothalamic and pituitary disturbance, at 28 years giving ventricular obstruction requiring short-circuit operation and dying at 32 years with loss of vision, behaviour disorders, epilepsy, and finally bilateral adrenal hæmorrhages. No radiation therapy was given.

CASE REPORTS

Case 1 (M.S., Female, No. 45/222).—Born with café-au-lait patches scattered over her skin and a congenital pseudo-arthrosis of the right tibia, she developed a central proptosis of the right eye at the age of one and a half. One year later when first seen the right eye was more proptosed and quite blind; there was bilateral optic atrophy and an enlarged right optic canal radiologically. She was submitted to two operations. Initially the optic chiasm was explored below the right frontal lobe and the intracranial part of the right optic nerve and the chiasm were involved in tumour although the left nerve appeared normal. The right optic nerve was sectioned at the optic canal and at the chiasm and this intracranial portion removed, leaving the swollen, tumorous chiasm behind. One week later the severely proptosed right eye was enucleated and the remainder of the right optic nerve removed from the orbit and canal. She was last seen recently at the age of 18 years, supporting her parents with her earnings. The pseudo-arthrosis had united without deformity following operation 10 years ago. *Her visual acuity remains J 1 in the left eye and the visual field full.* Radiography showed the sella and the left optic canal to be normal.

The pathological examination confirmed that the posterior line of section at the chiasm had passed through tumour. Microscopically it was a typical piloid glioma of the optic nerve with Rosenthal fibres. The tumour reached the right globe but did not invade it. In spite of an incomplete removal there is no evidence of advancement after 16 years.

Case 2 (J.R., Female, No. 297679).—She was born with café-au-lait patches and even at the age of 2 years was thought to have defective vision. The sight appeared to deteriorate slowly as she grew, and from the age of 3 years she increased her weight to beyond normal limits. When first seen at 10 years of age she had 'pituitary-like' obesity

and weighed 7½ stone (50 kg.). She had no proptosis but bilateral optic atrophy, and visual acuity was reduced to finger-counting in the left and J.20 in the right eye. The visual fields showed concentric constriction. An air encephalogram failed to outline a normal anterior end of the third ventricle and the air did not pass further forward in the basal cisterns than the prepontine cistern.

The chiasm was explored below the right frontal lobe and found to be swollen with the intracranial portions of the right optic nerve 7 mm. and the left 12 mm. in diameter. They were grey and lustreless; an attempt at biopsy was unsuccessful. She was last seen recently at the age of 17 years, earning her living in an office. She was slightly overweight with normal distribution of body hair but not yet menstruating. *Her visual acuity had apparently improved to J.10 on the right and J.20 on the left.* Both optic disks remained atrophic. X rays of the optic canals showed no enlargement, and an X ray of the sella was normal. Although there was no histological verification the chiasm and nerves were seen and there was little doubt about the clinical diagnosis. She received no treatment and her vision appeared to improve spontaneously over the 7 years she was under observation.

Case 3 (M.B., Male, No. 46/394).—He first complained of some failure of vision at the age of 38 years, and four years later was found to have bilateral optic atrophy and visual acuity of less than J.20 in each eye. Radiologically the right optic canal was slightly enlarged and the sella was gourd-shaped. An air ventriculogram suggested an irregular defect in the anterior third ventricle. The chiasm was explored below the right frontal lobe and found to be 2 cm. in thickness with the right nerve three times and the left nerve twice the normal thickness. A small biopsy was taken from the right nerve but nothing else was done. He was seen recently by his own practitioner. Aged 56 years, he was working as a farmer, with his vision having *improved* to J.6 on the left and J.18 on the right. He was unable to attend for radiological examination due to the pressure of work. The pathological examination showed a typical piloid glioma of the optic nerve with Rosenthal fibres and without any special distinguishing features. It is not known how long this tumour had been present, but there were bony changes in the sella at the age of 42, and 14 years later little change in his clinical condition. His vision had at first deteriorated and then spontaneously improved.

Case 4 (J.S., Female, No. 59/12479).—She was born with café-au-lait patches like her mother and placed in a foster home when aged 2 years. At that time she had a left divergent strabismus, but not until she was 7 years of age was it discovered that the left eye had no perception of light and that she had bilateral optic atrophy. Her right vision was J.1 6/6, in spite of an atrophic disk. There was no proptosis and the visual field was full. The left optic canal was twice the size of the right but the sella was normal. An air encephalogram showed a normal anterior wall of the third ventricle and a normal cisterna chiasmatis. The chiasm was explored below the left frontal lobe and found to be twice the normal size, and the intracranial portion of the left optic nerve was similarly thickened to the foramen of the optic canal. The right nerve appeared normal. The left nerve was sectioned at the chiasm and at its entry into the orbit (after removal of the roof of the optic canal) where it was seen to be of normal thickness. She was last seen recently at the age of 10 years developing normally and doing well at school, with the right visual field and acuity as before, and the disk remaining pale. X ray showed the right optic canal to be still normal and no change in the sella. The posterior line of section passed through tumour. Microscopically it was a typical piloid glioma of the optic nerve with nothing remaining of the normal nerve architecture.

Case 5 (J.F., Female, No. 290361).—She first noticed slight proptosis of the left eye at the age of 27 years, and when seen one year later complained of visual loss in the left eye (to finger counting only) which she had just discovered. On examination there was slight proptosis of the left eye with an irregular inferior altitudinal field defect and papillœdema. She had no signs of von Recklinghausen's disease. Radiologically there was no abnormality in the orbit or sella. The right eye was normal in all respects. As there was some doubt about the diagnosis an orbitotomy was performed by the Dickson-Wright approach (Stallard, 1958) and a biopsy taken from the thickened convoluted optic nerve which appeared to double back on itself in the orbit. After the pathology had been confirmed a second operation was performed for total removal of the left optic nerve. A left frontal osteoplastic flap was turned and the chiasm explored below the left frontal lobe. The chiasm and intracranial portion of both optic nerves appeared normal. The left optic nerve was then sectioned at the level of the chiasm and the optic canal and orbit were unroofed. By turning down once more that small hinge of bone which formed the lateral portion of the supra-orbital ridge, and had been first turned down at the Dickson-Wright orbitotomy, it was possible to expose the whole orbital contents and remove the optic nerve *in continuity* up to the globe. The globe was left in situ to accord with the wishes of the patient, expressed before the operation, and it was necessary to dissect tumorous nerve from the back of the sclera. She was last seen recently at the age of 34 years with no evidence of recurrence. The left orbital contents were sunken and the left cornea opaque, so that the fundus could not be seen. The right eye remained normal in all respects. The sella and the optic canal on the right were normal radiologically. Since operation she had given birth to her second quite normal child.

The pathological report showed normal nerve at the posterior line of section. The nerve at the anterior line of section from the globe was involved in tumour. Microscopically the tumour was a typical piloid glioma of the optic nerve. Removal was considered to have been complete by virtue of the clean dissection from the globe and it had not recurred after 7 years.

Case 6 (L.E., Female, No. 54/5958).—She was born with numerous café-au-lait patches. A mild proptosis of the right eye developed at the age of 18 months. At the age of 2 years she was investigated for this and was found to have bilateral papillœdema. Neither the visual acuity nor the visual fields could be tested satisfactorily. Both optic canals were enlarged radiologically, but the sella appeared normal; an E.E.G. was normal. At operation there appeared to be raised intracranial pressure with flattened gyri. The chiasm was explored below the right frontal lobe and found to be swollen, and both optic nerves coming from the chiasm appeared like spring onions in contact with each other until they entered their canals. The right frontal lobe was tough and unyielding, and on attempting to aspirate the lateral ventricle the right frontal horn could not be found; it was obviously not enlarged and third ventricle obstruction not the cause of the raised pressure. A tough resistance in the white matter of the frontal lobe was felt, suggesting a glioma in that position also, but a biopsy of the frontal lobe showed no abnormality. At the age of 3, one year after the first operation, both disks were flat and pale, but the right eye, which was by this time more proptosed and beginning to ulcerate, had to be removed together with the orbital part of the right optic nerve which was involved with tumour. She was last seen recently at the age of 10 years developing normally and doing well at school. Her visual acuity in the remaining eye was J.2 and the disk showed fairly severe secondary optic atrophy. The sella remains normal but the optic canals were both three times the normal size.

Pathological examination of that part of the tumour removed showed the globe free of tumour at the disk. The posterior line of section through the nerve had passed through tumour. Histologically it was a typical piloid

glioma of the optic nerve with Rosenthal fibres and complete destruction of the normal structure of the nerve.

No explanation was found for the papilloedema or the apparently raised intracranial pressure at the first operation, the difficulty in finding the ventricle, or the indurated frontal lobe. It is thought that it may have been an episode of some form of local body reaction to the presence of the tumour, for it subsided after one year without any surgical interference with the tumour.

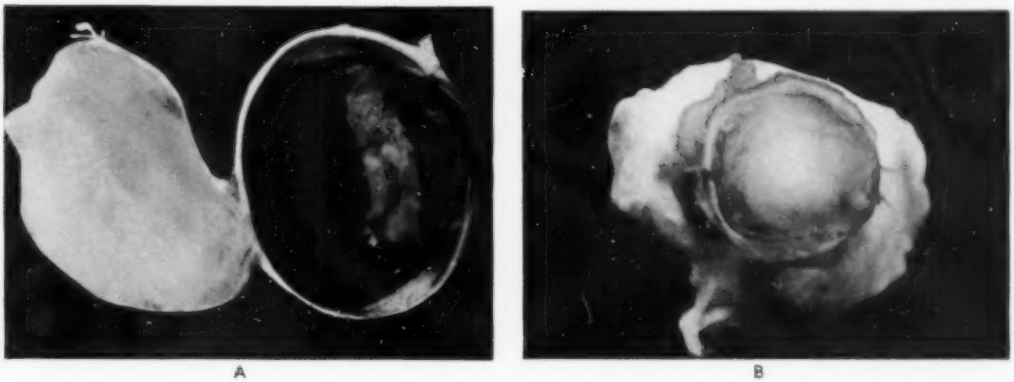
Case 7 (D.B., Male, No. 54/30683).—Born with a few café-au-lait patches on the skin, he developed left proptosis at 18 months. At the age of two the left eye was quite blind and the disk previously seen under general anæsthetic elsewhere had been described as slightly swollen with dilated veins. The left optic canal was enlarged radiologically to twice the size of the right. The chiasm was first explored below the left frontal lobe, and although the left nerve was twice its usual size at its entry into the optic canal it was of normal width at the chiasm. This intracranial portion of the nerve was removed but at the chiasm where it was cut some tumour could be seen by the naked eye to have been left behind. However, it was decided not to section the chiasm. Six weeks later, and through an orbitotomy approach, the globe and remainder of the tumorous optic nerve were removed. Although the frontal bone flap was turned down in addition to increase the exposure, access to the canal was difficult.

He remained well for five years, and then, in a period of a few weeks, developed a soft painless swelling subcutaneously at the nasion with bone erosion of the anterior wall of the frontal sinus seen radiologically. After a preliminary needle biopsy the swelling was incised and a mass of soft unencapsulated pinkish-grey tumour material was incompletely sucked from an extensive cavity replacing the ethmoidal sinuses. He recovered well from this and when seen recently 12 months after this third operation the wound was well healed and the tumour showed no signs of reforming. The vision in his right eye remained J.1; his optic disk could not be seen due to lack of co-operation but his visual field appeared full. Radiography showed no further bone destruction and the right optic canal and sella were normal.

Histologically the tumour of the nerve was a typical piloid glioma and it was thought that tumour cells could be seen at the level of the posterior section from the chiasm as was suspected at operation, and removal from this site was incomplete. The histology of the tumour material removed from the ethmoidal sinuses five years after removal of the eye and nerve was difficult to interpret; the small amount obtained from the needle biopsy suggested a sarcoma, but when more was obtained at open operation it appeared to be a piloid glioma growing freely in an empty space and the cells were loosely arranged amidst a network of capillary blood-vessels.

Case 8 (P.McG., Female, No. 54/33113).—She was born with café-au-lait patches over her skin, but was well until 5 years of age when she developed a left proptosis over a period of 6 weeks. On investigation her visual acuity could not be ascertained although she was not completely blind in the left eye in spite of a primarily atrophic optic disk. The optic canals and sella were normal radiologically and an air encephalogram showed a normal anterior wall of the third ventricle. The right eye was normal in all respects. In the hope that this tumour could be completely removed, the chiasm, optic nerve, and globe were exposed by a single orbitofrontal bone flap and removal of roof of the orbit and optic canal (*see Figs.* 357–360). The chiasm was normal and the posterior line of section passed through normal nerve just anterior to the chiasm. The tumour was confined to the orbital portion of the nerve but the whole nerve and globe were removed in one. She was last seen recently at the age of 12 years, developing normally with a healthy socket. There was no evidence of any disease in the right eye and the right optic canal was normal radiologically.

Pathologically the tumour was a typical piloid glioma of the nerve without invasion of the globe and with the posterior end of the nerve free from any suspicion of tumour cells (*Fig.* 355).



A B

FIG. 355.—A, Section of the globe and part of the tumorous nerve of *Case* 8, showing the spread of the tumour into the dural sheath of the nerve and the bulky fibrous reaction produced. The retina and globe were not invaded. The great thickening of the nerve in the orbit produces marked proptosis and occasionally papilloedema by venous obstruction. B, A cross-section of the intra-orbital portion of the optic nerve in *Case* 8, showing the reaction produced in the dural sheath by the outward spread of piloid glioma cells from their origin in the nerve.

Case 9 (J.A., Female, No. 38/208).—She was born with café-au-lait patches, but was otherwise well until the age of 7 years when she had difficulty with vision. She was found to have bilateral optic atrophy and visual acuity of J.8 on both sides; she had precociously developed small breasts and pubic hair but had not menstruated. Both optic foramina were enlarged and the floor of the sella was depressed radiologically. There was no increase of intracranial pressure when she was first seen. No operation was performed. Four years later when aged 13 years she was reported to have become almost totally blind, hypersomnic, and had developed frequent attacks of petit mal. She died in coma and her brain was sent for post-mortem examination. The tumour was found to occupy the interpeduncular space and to obliterate completely the chiasm. It had blocked the foramen of Monro

and produced severe internal hydrocephalus. The optic nerves were seen merging into the tumour mass. The histology shows a typical piloid glioma of the optic nerve and chiasm with Rosenthal fibres.

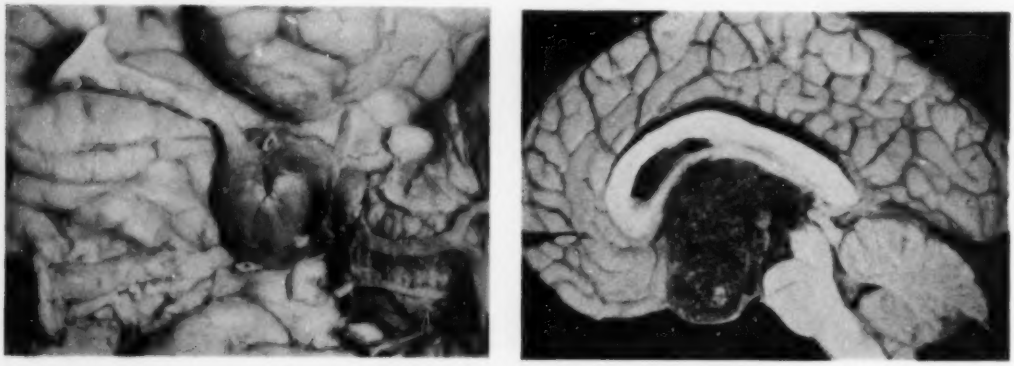
Case 10 (K.S., Male, No. 112455).—He first noticed failing vision in his left eye when aged 19 years. He was investigated one year later and the only physical signs were bilateral optic atrophy with acuity on the left J.18 and on the right J.2, with an irregular temporal hemianopia in the right field. He had no signs of von Recklinghausen's disease. Radiologically the dorsum sellæ and the posterior clinoids were eroded, suggesting a fairly extensive tumour in the region of the third ventricle, but the optic canals were normal in size. An air encephalogram showed a filling defect in the anterior part of the third ventricle. The chiasm was explored and found to be three times normal size and the commencement of each optic nerve was swollen. No biopsy was taken.

He re-attended at the age of 28 years with severe hypersomnia. He was now married and had one child. His left eye could just perceive light, and the temporal hemianopia in the right was now complete although the acuity remained J.1. Both disks were pale and atrophic. A myodil ventriculogram was performed and the myodil would not leave the right ventricle due to foramen of Monro obstruction by the upward extension of the tumour. Following this procedure he became hypothermic and hypotensive and was given cortisone. Soon afterwards he developed papillœdema due to complete ventricular obstruction and a bilateral ventriculo-cisternostomy (Torkildsen's operation) was performed. He made a fair recovery and returned home to an office job. At the age of 31 years he developed 'grand mal' epilepsy which was partly controlled by anti-convulsants. His weight had increased by 9 kg., he was impotent, and the hypersomnia had increased. One year later, at the age of 32 years, he deteriorated mentally with periodic violent behaviour, and he lost his job. At this stage his right visual acuity was J.4 with only the upper nasal quadrant of the right visual field remaining. His left eye was quite blind. He deteriorated and died suddenly the same year in a chronic-sick hospital from bilateral adrenal haemorrhages.

His brain, optic nerves, and posterior portions of both globes were examined post mortem (*Fig.* 356). The optic nerves emerged from a tumour mass which engulfed the chiasm and except for the first few millimetres were free from tumour both to the naked eye and histologically. The tumour mass had replaced the floor and walls of the third ventricle and spread throughout the ventricular system to the fourth and lateral ventricles. The pituitary stalk was attenuated and merged from a normal gland into the tumour mass. Histologically the tumour was a typical piloid glioma with Rosenthal fibres. Where it was growing freely in the lateral and fourth ventricles the cellular architecture was more loose and similar to that seen in *Case* 7.

DISCUSSION

As one purpose of this paper is to record the progress of these cases it is hoped that other centres will record the natural history of their cases of piloid gliomas of the optic nerves and chiasm that have been followed for two decades or more. Until a reasonable knowledge of the expectation of life and morbidity accompanying these tumours has been gained it will always be difficult to assess the indications for and the efficacy of treatment by any method.



A B

FIG. 356.—A, A view of the base of the brain and B, a sagittal section of the brain to show the piloid glioma of the chiasm in *Case* 10 (*see* text).

In this series, of the 9 tumours examined histologically the cyto-architecture was essentially identical. In 2 cases, *Cases* 7 and 10, there were parts of the tumour extending into a cavity—the first into the nasal sinuses, the second into the lateral and fourth ventricles. In these situations the cyto-architecture was much more loosely formed than elsewhere but the characteristic cells were present. (The strange way in which the tumour traversed the bone of the frontal sinuses in *Case* 7 remains unexplained.)

Where the gliomas grew in the intra-orbital portion of the optic nerve and expanded into the dural sheath covering, the fibrous tissue elements proliferated, giving firmness and increased bulk to these parts of the tumours, and the cells were seen to be more densely arranged (*Fig.* 355).

In the intracranial parts of the optic nerves and in the chiasm the tumours had less fibrous reaction and in the early stages were less in bulk with a preponderance of tumour cells.

There was nothing histologically to indicate the possible rate of growth and no means of distinguishing the histology of the tumours which proved fatal from those more benign. Histology, therefore, gave no help in prognosis.

There was nothing in the early clinical features of the 2 fatal cases to distinguish them at the outset

FIG. 357.—To show the skin incision for the combined orbitofrontal osteoplastic bone flap. The horizontal portion is taken through the eyebrow and is thus concealed.

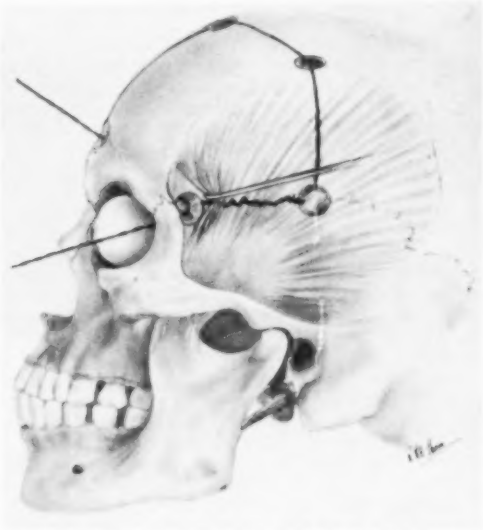


FIG. 358.—The low anterior burr hole is made in the temporal fossa and opens into the anterior cranial fossa above and the orbit below. The Gigli saw is passed first from the lowest, midline burr hole cut into the temporal fossa and then throughout the lower opening into the orbit after preliminary stripping of the orbital periosteum.

FIG. 359.—The lowest saw cut has been made through the orbital margin and the roof of the orbit. The Gigli saw is next taken back through the orbit to the temporal fossa and a cut is made through the frontal process of the zygomatic bone just below the zygomaticofrontal suture.

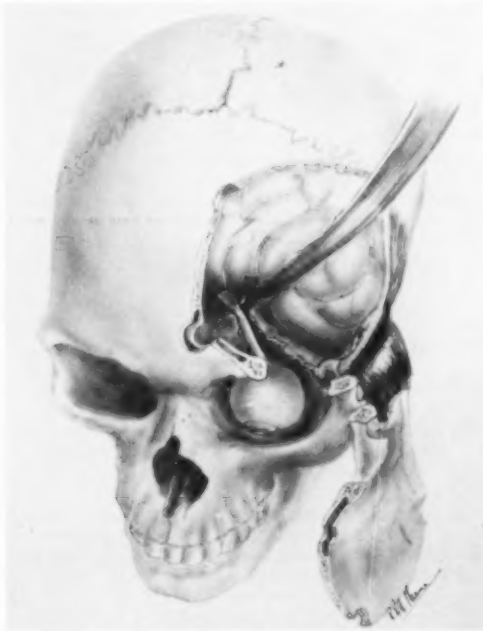


FIG. 360.—The combined orbitofrontal osteoplastic bone flap has been turned down, hinged on the temporal muscle. The roof of the orbit and the roof of the optic canal have been removed to allow the full length of the optic nerve to be exposed and removed from the chiasm to the globe. The frontal air sinuses have been opened in this case but can be sealed at the end of the operation with a fascial graft.

from those who are still alive and apparently have more benign tumours.

Episodic growth was suspected in *Cases* 6 and 7 and in the former there appeared a transient episode of increased intracranial pressure with papillœdema. This was found not to be due to ventricular obstruction and may have been a transient local brain reaction to the tumour.

Some of these tumours appear to have stopped growing (*Cases* 1, 2, 4, and 6), but they need to be followed for longer periods. Lundberg (1936), McFarland and Eisenbeiss (1950), and Traveras, Mount, and Wood (1956) all hint at the possibility of spontaneous cessation of growth but do not record any with long-term follow-up. It is thought unlikely that subtotal removal or biopsy will have induced cessation of growth or have influenced its rate.

Several cases have occurred in this unit of intracranial tumour with histology identical to these optic pathway gliomas, but where the tumour has been confined to the wall of the third ventricle, the anterior pole of the frontal lobe, or the sub-frontal region. The histology is also identical with the 'juvenile' type of cerebellar astrocytoma. With the exception of the tumour in the cerebellar hemispheres the cases did not conform to a clinical picture embracing age of onset, appearance at operation, or benignity of course (many being rapidly fatal). None was associated with von Recklinghausen's disease and none appeared to stop growing. The piloid gliomas of the cerebellar hemisphere, however, presented a typical clinical picture with onset usually in childhood, and often long survival after operation for release of pressure and complete (but possibly on some occasions incomplete) removal of the tumour. These cases were not usually associated with von Recklinghausen's disease. The question arises whether some of these tumours of the cerebellum are self-limiting, stopping with the onset of puberty, and behaving rather like hamartomas.

It can be seen from this small series that the association of gliomas of the anterior optic pathways with von Recklinghausen's disease is not invariable. Like many rare malformations and rare tumours of the nervous system, these gliomas are commoner in, but not confined to, patients with von Recklinghausen's disease. They are perhaps the commonest tumours of the central nervous system in young children with von Recklinghausen's disease.

TREATMENT

The apparent benignity of some of the tumours must affect the choice of treatment. In some instances the choice is obvious. If total surgical removal is possible for a tumour confined to the nerve with a blind eye, then following operation one would have justification in hoping for permanent cure. Hudson (1912) quotes one case record where the patient was still alive after 24 years. The relief of ventricular obstruction by ventriculo-cisternostomy (Torkildsen's operation), the removal of a grossly proptosed and infected eye, and the verification of the diagnosis by inspection and biopsy are all surgical procedures that must, at times, be considered necessary in their management.

Difficulty arises when total removal cannot be undertaken because the tumour involves the chiasm and wall of the third ventricle or because total removal would mean the sacrifice of useful vision. Martin and Cushing (1923), Mehney (1936), and Fowler and Matson (1957) have resected through the chiasm and optic tract where only parts of these structures appeared to be involved in tumour, in an attempt to perform a complete removal. A clear margin of 1 cm. beyond swollen, tumorous tissue was not taken so that the removal, always difficult and dangerous in this situation, was probably incomplete and the procedure unjustifiable. As seen in *Case* 7 and in a case described by Fowler and Matson (1957) tumour cells were thought to extend for at least 1 cm. beyond the posterior limit of a swollen portion of the optic nerve, so for a complete removal at least this margin should be taken. There is no reason to suppose nor evidence to support a belief that partial removal affects the growth of the remainder.

Too few case reports state whether or not the posterior line of section was free from tumour cells when claiming complete removals, but, as Verhoeff (1932) pointed out, to recognize the difference between glioma cells in the nerve and an increase of innocent glial cells that occur in response to the partial atrophy of the compressed nerve-fibres may be impossible.

Effective surgical palliation by restoration of some vision is said to have followed decompression of swollen, tumorous nerves, by unroofing the optic canal or by incising the tumour sheath (Davis, 1939), or by intracapsular removal of an intra-orbital tumour (Craig and Dodge, 1955). Decompression would therefore appear to be a useful procedure but vision is sometimes improved spontaneously in time (*Cases* 2 and 3).

Unless there is clear ophthalmoscopic evidence of optic disk or retinal involvement, the globe can probably be left for cosmetic reasons after the nerve has been sectioned at the sclera. It can be removed later if there is any suspicion of recurrence in the eye (Davis, 1940, and *Case* 5).

Cushing (1930) was the first to conclude that surgery was contra-indicated once it was certain that the glioma involved the chiasm and was therefore inoperable. He hoped that in the future radiotherapy would be the treatment of choice. Levitt (1937) advised radiotherapy as the best treatment but gave no details. The greatest advocates have been Traveras, Mount, and Wood (1956) who described 34 cases treated primarily by radiation and went on to claim 3 significant results. First, they claimed that radiotherapy would produce shrinkage of an intra-orbital glioma of the optic nerve with complete resolution of proptosis, in some cases lasting several years. Secondly, it would induce the shrinkage of gliomas of the chiasm and this could be watched by the altering outline of the third ventricle seen on serial air encephalograms. Thirdly, they claimed lasting improvement in vision and reduction of papillœdema in some of the cases treated by radiotherapy only. The details given in their paper, however, do not allow one to give unequivocal credit to the X rays for the subsequent well-being of the patient or the improvement in vision, in spite of their claims. In at least one spectacular case improvement in vision might have been attributed to the

surgical relief of raised intracranial pressure alone. Of the 3 cases given radiation for proptosis, 1 of whom lived for twenty-two years without evidence of recurrence of the proptosis, none was verified surgically or histologically. The 3 cases with large chiasmal tumours giving rise to ventricular obstruction and requiring surgical by-pass operations were given radiation therapy in addition, but none survived for more than five months after the operation. Fowler and Matson (1957) found no improvement following radiation therapy in 2 of their cases with large chiasmal tumours.

In this series, in *Cases* 2 and 3, vision has improved over several years without the benefit of either surgical or radiation therapy, but proptosis when it has occurred has been relentless and has usually led to surgery.

The optimum course of radiation therapy is still under review by those who favour it, but in view of the natural history of the tumour that is slowly being unfolded the place of this form of treatment in its management is not yet proved.

SUMMARY

Ten cases of piloid glioma of the anterior optic pathways are described and the literature is reviewed. The cases illustrate, amongst other things, that the natural history of some of these neoplasms is prolonged, and that some tumours appear to behave as though they were completely benign. The slow growth and occasional spontaneous regression of symptoms make the assessment of surgical and radiation treatment difficult. Surgery is advocated if the tumour is confined to the optic nerve, and total removal can be effected. A combined orbitofrontal exposure is described which enables the whole length of the optic nerve to be removed in continuity. The benefits gained by partial surgical removal of these tumours are questioned, especially when useful vision is sacrificed, and operations on the optic chiasm and optic tracts are considered unjustifiable. Many results claimed for radiation therapy are not considered to be proved, and none of the present 10 cases was so treated. The irradiation of the part of a tumour within the orbit may reduce proptosis and prevent the loss of the globe. A long and detailed follow-up of a larger number of histologically verified cases is necessary for a more consistent picture of the natural history to emerge.

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VERRUCOUS CARCINOMA PRESENTING AS MANDIBULAR OSTEOMYELITIS

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THE existence of a peculiar slow-growing proliferative carcinoma of the oral epithelium, having a relatively good prognosis, was first suggested by Blair, Moor, and Byars (1941). This tumour was called a 'papillomatous epithelioma'. The features described are similar to those reported by Ackerman (1948) in his account of 'verrucous carcinoma of the oral cavity'. This title was reserved for an epithelial tumour having clinical, pathological, and prognostic features different from other intra-oral carcinomas. Similar tumours were described by Friedell and Rosenthal (1941), but no other account appears in the available literature.

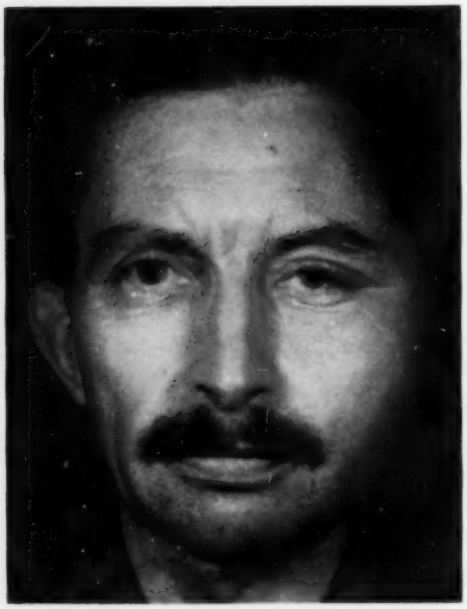

FIG. 361.—*Case* 1. A 40-year-old man with a large brawny swelling of the left cheek and infratemporal region. This swelling had been present for 7 months following the extraction of molar teeth.

Ackerman and Johnson (1952) examined 203 oral carcinomas and found 37 examples of verrucous carcinoma. Most of these tumours occurred in elderly males, many of whom were tobacco chewers. Twenty-nine originated on the buccal mucosa or lower alveolus, 7 on the hard palate or upper alveolus, and there was 1 lesion of the tongue. Growth was slow, but if untreated there was extension over a wide area to form a papilliferous mass. Leucoplakic changes were frequently seen at the margin of the tumour. Deep penetration was usual, with perforation of the cheek, fixation to periosteum, and, later, erosion of the mandible. Palpable cervical lymph-nodes were found, but in 10 block dissections only 1 node showed secondary tumour. Clinical or pathological evidence of distant metastases was not seen. Microscopically these tumours showed a papillary structure with deep penetrating processes of extremely well-differentiated squamous epithelium. The basement membrane remained intact according to these authors.

A recent opportunity of examining material from a patient with an undoubted verrucous carcinoma prompted a search for similar cases treated at Hammersmith Hospital during the past twenty-five years. Biopsy and resection specimens, some with block dissections, were available for review in 177 cases of oral carcinoma. Sixty-four of these tumours were situated on the buccal mucosa or lower alveolus, where Ackerman found most of his verrucous carcinomas, but the distinctive clinicopathological features of this neoplasm could be recognized with certainty in only 3 cases. All were tumours of the lower alveolus.

FIG. 362.—*Case* 1. Radiograph taken 7 months after extraction of lower second and third molars, showing a radiolucent area, a retained root, and erosion of the base of the coronoid process.

It is the purpose of this paper to report the unusual mode of presentation common to all three examples of this little-known variant of oral squamous carcinoma.

CASE REPORTS

Case 1.—September, 1956: A man, aged 40 years, attended his dentist for the surgical removal of the horizontally impacted lower left third molar. To facilitate access the second molar was sacrificed. Post-operative swelling persisted and severe trismus followed.

April, 1957: Referred to hospital with a tender brawny swelling which extended from the zygomatic region above, to the inferior border of the mandible below (*Fig.* 361). The anterior teeth could be separated only a few millimetres. The lower left buccal sulcus was obliterated posteriorly and there was a cavity at the extraction site.

Radiographic examination showed the unhealed socket, with further erosion of bone, but no sequestrum formation (*Fig.* 362). The mesial root of the second molar was still present. A clinical diagnosis of chronic osteomyelitis of the mandible was made. External drainage on two occasions and antibiotic therapy did not relieve symptoms. The drainage wound at the angle of the mandible failed to heal. Extraction of the first molar and the root of the second molar, with saucerization of the affected area, resulted in a slight improvement. No bacteriological or histological evidence of actinomycosis or tuberculosis was found.

November, 1957: An abscess cavity extending from the posterior end of the alveolus, into and deep to, the masseter was opened. This abscess contained greenish-yellow caseous material from which *Streptococcus viridans* was cultured. Papilliferous processes, the first suggestion of neoplasia, were found in the wall of this cavity. A few weeks later there was radiographic evidence of almost

FIG. 363.—*Case* 1. Photomicrograph showing epithelial hyperplasia without evidence of malignancy. This was found in many early biopsies. H. and E. (× 30.)

complete reconstruction of the alveolus, but erosion of the coronoid process and zygomatic arch was now seen. Subsequently drainage of abscesses in the infratemporal fossa and the submandibular triangle became necessary.

September, 1958: A fungating papilliferous tumour now appeared through the mouth of the infratemporal sinus. This grew rapidly, reaching a size of 60 × 50 mm. On the basis of the current biopsy, the tumour, adjacent muscles, parotid and submandibular salivary glands, zygoma, and the posterior half of the left mandible were excised.

April, 1959: A tumour mass was found in the left side of the neck and block dissection was carried out. This was followed by a right block dissection in November, 1959.

December, 1959: The patient died three years and three months after the onset of symptoms.

PATHOLOGY: Biopsies taken from the oral mucosa and the mouths of sinuses are difficult to interpret. These show hyperplastic epithelium with marked acanthosis, producing broad, blunt epithelial downgrowths. Stratification is normal and the basal layer is intact (*Fig.* 363). In several biopsies the subepithelial connective tissue has the appearance of granulation tissue, rich in lymphocytes and plasma cells. The true pattern of the

FIG. 364.—*Case* 1. Photomicrograph showing folded epithelial papillæ. The clefts between the papillæ are filled with keratin and parakeratotic squames. H. and E. (× 4·1.)

lesion is seen in the biopsy of the proliferative infratemporal tumour (*Fig.* 364). This shows a folded, papillary structure with marked surface parakeratosis. Normal stratification is preserved. Deep clefts between the folds are filled with voluminous parakeratotic squames. The rete cells show some variation in size and shape but mitoses are few. Blunt epithelial processes extend deeply into underlying muscle, but there is no invasion through the basal layer. The vascular stroma is infiltrated with inflammatory cells.

The cut surface of the excised tumour (*Fig.* 365) shows broad white strands, infiltrating deeply into underlying muscle. In the deeper areas the tumour fingers form a tortuous pattern.

The two tumour masses found in the block dissections do not show any recognizable lymphoid tissue. One is a keratin-filled cyst with an incomplete epithelial lining. The continuity of the basal layer is broken at one point. The second mass is an undifferentiated carcinomatous nodule (*Fig.* 366). None of the fourteen lymph-nodes examined shows deposits of tumour.

At autopsy, performed elsewhere, extensive fibrous scarring in the left face and neck was said to be free of recurrent tumour. Lymph-node and distant metastases were not found.

Case 2.—July, 1951: A man of 42 years, who had been edentulous for 20 years, complained of a painful swelling in the right submandibular area, which extended upwards on to the lateral surface of the mandible. Intra-oral examination showed an intact oral mucosa but there were fluctuant swellings buccally and lingually in relation to the lower right alveolar process. The lesion appeared to be an inflammatory condition, but no obvious cause could be found. Drainage via a submandibular incision released a copious flow of pus. Later, infected granulations and necrotic material were curetted from the buccal surface of the mandible. A discharging submental sinus remained.

December, 1951: Pain, swelling, and discharge returned, and the right side of the mandible was again exposed. Much of the body and the whole of the ascending ramus were found to be involved in an infective process. The mandible was divided through the lower left canine region and the right half, including the condyle, was excised. External healing was never complete and intra-oral sinuses appeared over the remaining part of the mandible.

August, 1952: At a further operation to repair the defect, dissection in submandibular fibrous tissue demonstrated multiple cysts and epithelial tracts. An operative diagnosis of adamantinoma was suggested.

March, 1953: The patient was referred to Hammersmith Hospital for the removal of the supposed adamantinoma, which had now recurred in the floor of the mouth. part of the mandible, together with a right block dissection, was carried out. Reconstructive procedures were started one year after this extensive local resection.

1957: There were no clinical signs of local recurrence or distant metastases when the patient died of bronchopneumonia, six years after first noticing symptoms. No autopsy was performed.

FIG. 365.—*Case* 1. A, Cut surface of recurrent tumour in the infratemporal region, showing proliferative growth with deep extension into underlying muscle. B, Verrucous tumour above and mouth of sinus below, seen from the skin surface.

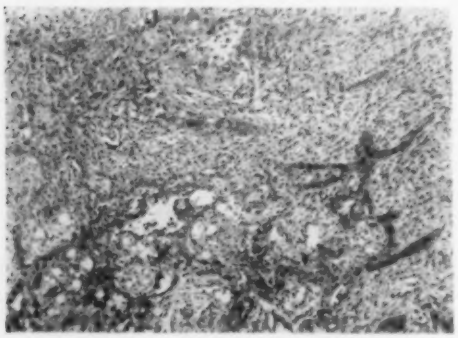

FIG. 366.—*Case* 1. Photomicrograph showing strands of undifferentiated squamous cells, not now producing keratin. H. and E. (× 60.)

FIG. 367.—*Case* 2. Photomicrograph showing clefts filled with keratinizing cells. The basal layers are joined by fine prickle-cell bridges. H. and E. (× 21.)

At operation there was extensive spread of tumour into the base of the tongue, the left anterior faucial pillar, and downwards to the soft tissues about the epiglottis. In parts there were multilocular cysts, containing grey flaky material which was thought to be keratin. Enlarged lymph-nodes were present on the right side of the neck. Radical excision of the tumour, tongue, and remaining

PATHOLOGY: Many early biopsies are as inconclusive as in *Case* 1. There are cup-shaped masses of hyperplastic squamous epithelium and some keratin-filled squamous-lined cysts. The most striking feature is the depth of epithelial penetration, without evidence of disruption of the basal layer. There is nothing to support the clinical diagnosis of adamantinoma.

The excision specimen showed tumour ulcerating through a submental skin ellipse. There were nodules, showing central necrosis, at the base and on the dorsum of the tongue. Microscopically the tumour shows a verrucous surface with long penetrating folds of very well-differentiated epithelium. Clefts, some empty and some

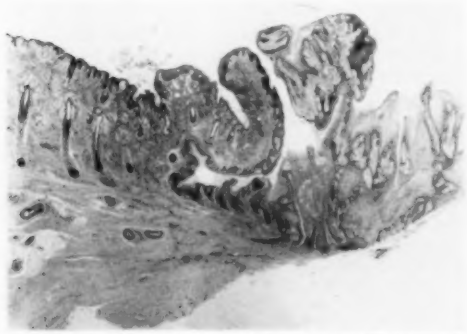

FIG. 368.—*Case* 2. Photomicrograph showing verrucous carcinoma from the mouth in continuity with skin epithelium. This perfect fusion was an unusual finding. H. and E. (× 4·1.)

FIG. 370.—*Case* 3. Photomicrograph showing an island of keratin with inflammatory cells and a giant-cell reaction in the stroma. The cleft above is lined by a thin squamous layer and is filled with inflammatory cells. H. and E. (× 23.)

filled with eosinophilic debris, are present. The deeper prolongations of the epithelial processes are joined by fine prickle-cell bridges, only two or three cells wide (*Fig.* 367). There is fusion of tumour epithelium with submental cutaneous epithelium, which, in this one section, suggests an apparent extra-oral rather than intra-oral origin (*Fig.* 368). The lymph-nodes are free from metastases.

Case 3.—1949: An 84-year-old man attended his dentist for the extraction of an upper left molar. At that time a small, symptomless swelling in the lower left third molar region was noticed. In view of the advanced age of the patient no treatment was advised.

June, 1953: The patient presented with a painful swelling at the left angle of the mandible. Multiple sinuses were discharging pus into the mouth. Radiographic examination showed the presence of an unerupted lower third molar with rarefaction of the surrounding bone. A diagnosis of osteomyelitis was made. The third molar was extracted and the infected area of the mandible was saucerized. The intra-oral wound failed to heal.

August, 1953: The patient was readmitted with a large brawny swelling of the left cheek and upper neck, which was pointing externally. There was ulceration in the buccal sulcus. Drainage of the abscess released a considerable quantity of pus.

At this stage he was referred to Hammersmith Hospital. Externally there was a discharging sinus, with heaped-up edges, at the lower border of the mandible

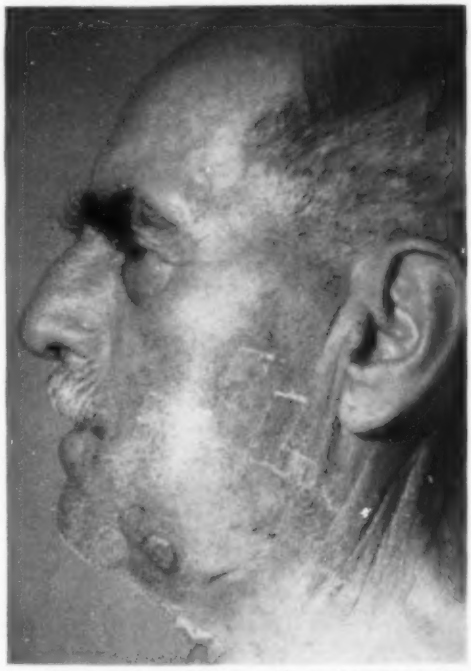

FIG. 369.—*Case* 3. An 84-year-old man with a discharging sinus at the lower border of the mandible. The mouth of the sinus is thickened and rolled.

(*Fig.* 369). Oral examination showed thickening and flattening of the alveolus, distal to the first premolar. There were several discharging sinuses and some ulceration of the cheek. A cervical lymph-node was palpable. Intra-oral biopsy at this stage showed the presence of squamous carcinoma.

At operation the tumour extended through the thickness of the cheek and upwards on the medial side of the mandible, to the level of the neck of the condyle. Wide local excision, including the left jaw and half the parotid gland, together with left block dissection, was carried out.

April, 1954: At a plastic operation tumour was seen in the region of the pterygoid muscles. Three small nodules on the skin of the neck were biopsied.

August, 1954: Extensive local recurrence of tumour gave rise to fatal hæmorrhage. No autopsy was performed.

PATHOLOGY: The resection specimen showed a track of white tumour extending from the oral ulcer to the mouth of the external sinus. One lymph-node below the angle of the mandible was enlarged. Microscopically this is a verrucous tumour as already described in *Cases* 1 and 2. A feature of this case is the tremendous stromal inflammatory change, with numerous groups of multinucleated foreign body giant-cells, adjacent to islands of keratin (*Fig.* 370). Many clefts are filled with inflammatory cells. Here well-defined epithelial stratification has been lost and the lining consists of a thin layer of flattened cells

(*Fig.* 370). The clinically enlarged lymph-node is the only one of 7 examined to show tumour deposits.

DISCUSSION

Verrucous carcinoma, as a distinct clinicopathological entity, has only been reported by one group of workers (Ackerman, 1948; Ackerman and Johnson, 1952; Ackerman and McGavran, 1958). In an attempt to find similar tumours in an independent series, 177 oral carcinomas were examined. Only the 3 cases described could be considered as definite examples of verrucous carcinoma. Three other tumours, 2 of the lower alveolus and 1 of the buccal mucosa, showed clinical and pathological features which suggested the diagnosis of verrucous carcinoma.

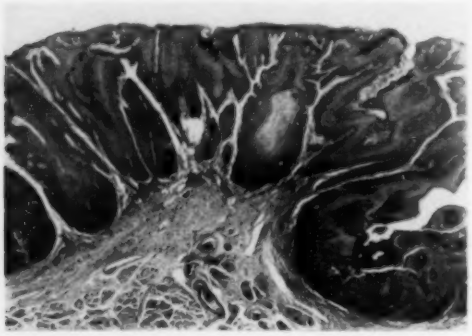

FIG. 371.—Photomicrograph of a verrucous lesion of the buccal mucosa showing marked epithelial proliferation without deep penetration. H. and E. (× 9.)

In these cases it was felt that the borderland between well-differentiated squamous carcinoma of conventional appearance and verrucous carcinoma had been approached, and they are not included in this report. Two other verrucous lesions of the buccal mucosa were seen. These showed extreme papillary epithelial hyperplasia, but there was no deep penetration (*Fig.* 371). These tumours probably represent verrucous carcinoma in its initial stages.

In Ackerman's series, verrucous carcinoma was found far more frequently than in the present group of oral carcinomas. Tobacco chewing was common in Ackerman's patients, but was not reported in the histories of the patients reviewed here. It was specifically excluded in one elderly male patient, who had a definite verrucous carcinoma (*Case* 3). This habit may have been responsible for the high incidence of verrucous tumours in Ackerman's report.

The clinical criteria laid down by Ackerman (1948) for the diagnosis of verrucous carcinoma are largely satisfied by the three cases reported. However, clinically verrucous tumour was seen only in *Case* 1, and even then not until almost two years after the onset of the disease. The single nodal metastasis (*Case* 3) and freedom from distant metastases in all cases are in keeping with Ackerman's findings. There was no histological proof that cervical tumour masses in *Case* 1 were situated in lymph-nodes. The survival time of these patients is difficult to assess, because the date of onset of symptoms due to the neoplastic process is uncertain. Their presentation as apparent inflammatory masses about the lower jaw, leading to a diagnosis of osteomyelitis of the mandible, does not appear to have been described before, but an atypical squamous carcinoma of the lower alveolus was reported by Radden (1951). This tumour had some resemblance to those described here, but a diagnosis of osteomyelitis was not considered.

The diagnosis of osteomyelitis is more likely to be made if there is precipitating local dental infection, as in *Cases* 1 and 3. In *Case* 2, where there was no such infection, osteomyelitis would have been unusual, as hæmatogenous infection of the adult mandible is far less common than osteomyelitis of dental origin (Wass, 1949). The clinical presentation of these tumours as osteomyelitis could be explained by stagnation and infection in the deeper parts of the crypts.

Microscopically, diagnostic difficulties are increased by the examination of superficial biopsies. Epithelial hyperplasia, hyperkeratosis, and inflammation of the corium are seen. These appearances may well be regarded as benign, or at worst may be interpreted as representing pseudo-epitheliomatous hyperplasia, in response to local inflammation. The diagnostic histological features of verrucous carcinoma, as discussed below, are only seen when large blocks become available.

A papillomatous surface, often heavily keratinized, is extensively folded to form deep clefts. If allowance is made for transverse and oblique sectioning of epithelial processes it can be readily imagined, but not proved, that the whole tumour is made up of a tortuously convoluted sheet of hyperplastic epithelium. The clefts seen in two dimensions can now be considered as three-dimensional crypts, filled with squamous cells, keratin, or pus. Masses of polyhedral parakeratotic squames, showing sharply defined borders and small, round, eccentric, and pyknotic nuclei, fill many of the crypts. A keratin plug marks the centre of other crypts. Normal stratification is preserved. Squames and keratin are being produced by orderly differentiation. Malignant dyskeratosis is not a feature of verrucous carcinoma. Infection of the crypts is well seen in *Case* 3, where polymorphonuclear leucocytes lie between processes which have lost much of their epithelial covering. The basal cells show slight variation in size and shape but few mitoses are seen. Epithelium is always sharply demarcated from corium, even about the fine bridges which pass between the deep extremities of the processes. Inflammatory cells collect in the corium in varying density but are most numerous where the crypts are infected. Keratin-filled cysts are seen, probably arising from the sequestrated ends of epithelial processes. If the lining of a cyst or crypt is lost the keratin excites a giant-cell reaction.

These histological appearances greatly resemble those of epithelioma cuniculatum of the foot (Aird, Johnson, Lennox, and Stansfeld, 1954). These authors considered this tumour to be peculiar to the foot, but they showed illustrations of tumours of the skin of the knee and the floor of the mouth which displayed extensive crypt formation. Fig. 293 of their paper is in fact from *Case* 2 reported here. The burrowing habit of epithelioma cuniculatum

has been reproduced by these three verrucous carcinomas, but the extremely debased epithelium (Aird and others, 1954) is not a feature of the oral tumours. Heavy keratinization of the normal surface, pressure, and venous stasis were thought to play an important part in the pathogenesis of epithelioma cuniculatum. Keratinization of the masticatory mucosa and the mucosa of the edentulous alveolus may be marked, but pressure and venous stasis are unlikely to be important in the pathogenesis of oral verrucous carcinoma. Pseudo-epitheliomatous hyperplasia is not uncommonly seen in the mouth in association with ill-fitting dentures or retained roots. It may be that these tumours started in this way before becoming carcinomas of the verrucous type. In *Case* 1 transition from hyperplasia to verrucous carcinoma and finally to undifferentiated carcinoma was seen. However, chronic irritation of the oral mucosa is common, and pseudo-epitheliomatous hyperplasia follows not infrequently. If verrucous carcinoma really had its origin in these changes it would be far less rare than this report indicates.

In conclusion it should be stressed that verrucous carcinoma is a slowly growing tumour which extends widely and forms multiple sinuses. An erroneous diagnosis of infection may be made. Superficial biopsies often fail to demonstrate the true features of the tumour, which are not seen until large blocks of tissue are examined. An awareness of this mode of clinical presentation of verrucous carcinoma, combined with early pathological diagnosis and treatment by wide local excision, may prevent a prolonged illness and fatal outcome in a tumour which metastasizes infrequently.

SUMMARY

1. Three examples of verrucous carcinoma were found in a series of 177 primary carcinomas of the oral epithelium. Clinicopathological data on these 3 patients are presented.

2. Clinically, these tumours presented as apparent inflammatory swellings in the mandibular region, suggesting a diagnosis of osteomyelitis. Slow but widespread local growth, with deep penetration, occurred. Only one lymph-node metastasis was found. Distant metastases were not seen.

3. The principal histological features of verrucous carcinoma are papillary growth, together with deep penetration by extremely well-differentiated squamous epithelium which shows normal stratification. Infection between papillary folds is probably responsible for the clinical presentation. Superficial biopsies fail to demonstrate the malignant nature of this tumour.

4. The pathogenesis of verrucous carcinoma is discussed.

Acknowledgements.—The patients whose case-histories are reported here were under the care of Professor M. R. Ewing of Melbourne, formerly of the Department of Surgery, Postgraduate Medical School, and Mr. A. K. Monro, also of the Department of Surgery. I am grateful to Professor C. V. Harrison and Dr. J. G. Azzopardi for their helpful advice and criticism during the preparation of this paper.

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EXPERIMENTAL SURGERY

THE USE OF THE UROFLOWMETER

(A PRELIMINARY REPORT)

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INTRODUCTION

ESSENTIALLY this instrument records the volume of urine passed per unit of time, simultaneously recorded with the fluctuations in the passage of voiding of this urine. This present study stems from the work of von Garrelts (1956, 1957 a, b), and the instrument used is a modification of that used by him.

A number of previous investigators have attempted to deal with the problem of describing in quantitative terms the flow of urine during the act of micturition. Ballenger, Elder, and McDonald (1932) measured the 'voiding distance' or 'cast distance' of the urinary stream and this method was also considered by Morales and Romanus in 1952 and 1954. Drake (1948) constructed an apparatus permitting a direct recording of the course of micturition. His apparatus consisted of a container suspended from a spring, the urine being passed into the container and the increasing weight of the container continuously recorded. Johanson (1953)

used a method of 'timed micturition' in which the length of time necessary to fill a 100-ml. container was recorded.

Von Garrelts collected the urine in a container, the changes in weight being transduced into electrical equivalents, which, after amplification, were recorded on a photokymograph. The recording apparatus is

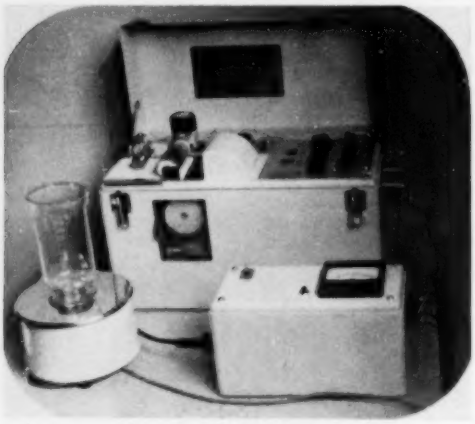

FIG. 372.—The uroflowmeter.

similar to an E.C.G. machine and functions in the same way. This is obviously the most accurate method and the instrument which we have used is a slight modification of von Garrelts's original model. (*Fig.* 372.)

The changes in pressure in the container are transduced, amplified, and are recorded simultaneously with the volume of urine voided per unit of time.

METHOD

Essentially, there are two recordings, an upper line which indicates the weight (volume) of urine passed per unit of time and a lower line which shows the variation in the rate at which that volume of urine is passed. The bottom line may be regarded as a differential of the top line. For convenience, an arbitrary standard has been maintained, i.e., a deflexion of one vertical centimetre corresponds with a hundred grammes. The bottom line indicates the differential rates of flow, so that 7 ml. are passed during a second early in the act of micturition whereas 20 ml. may well be passed at the height of the act. The minute variations seen on the lower line are emphatically not due to over-sensitivity of the instrument or to overswing. In fact, it is possible to record the passage of a single drop of urine on the lower line tracing. It is not possible to record such minute variations on the upper line, which must be regarded as an integration of the lower record. The patient micturates into a container under standard conditions, the tip of the penis being 4 in. above the upper edge of the container.

The great advantage of the method is that it is possible to record the volume of urine passed during *any given stage* of micturition. For example, in one normal individual 10 ml. were voided during the first second whereas 24 ml. were voided during the seventh second, this being the maximum rate. Other valuable information concerns the actual length of time required to evacuate the bladder; also it has been possible to plot out the curve of normal micturition and compare this with abnormal states such as urethral stricture and prostatic obstruction.

CONDITIONS

Study of Normal Micturition.—We have found that there is an initial rise in the rate of flow which reaches a plateau and then gradually declines. From the initial rise in the rate of flow it is possible to see that the volume of urine voided per second increases to a maximum, then remains fairly constant, finally declining. In other words, micturition can be divided into three parts—an initial rise, a middle plateau, and a final decline.

The amount of urine in the bladder influences the recording to a certain extent. The greater the volume of urine the greater the maximum flow per unit of time and the greater the rate of flow. For example, in a patient voiding 310 ml. the recordings were as follows:—

1st second, 1·7 ml.	
2nd second, 5·0 ml.	
3rd second, 9·0 ml.	
4th second, 11·5 ml.	Average voiding rate
5th second, 14·0 ml.	20 ml. per second
6th second, 16·5 ml.	
7th second, 19·0 ml.	
8th second, 21·0 ml.	

In a second normal patient voiding 520 ml. the figures were appreciably higher:—

1st second, 5·0 ml.	
2nd second, 11·0 ml.	
3rd second, 20·0 ml.	Average voiding rate
4th second, 24·0 ml.	24 ml. per second
5th second, 27·0 ml.	
6th second, 30·0 ml.	

Although the rate of flow varies with the volume of urine in the bladder the pattern remains essentially the same, provided the bladder content is not too small in amount.

Figs. 373, 374 show normal recordings.

Stricture of the Penile Urethra.—As with the normal subject there is a rise, a plateau followed by a terminal decline, but certain differences are immediately apparent.

1. The maximum volume passed per second at the height of micturition is considerably less than the normal range.

2. The initial rise is more gradual and the terminal decline more abrupt.

3. The oscillations of the lower line are exaggerated owing to the irregularity of the rate of flow.

Fig. 375 shows these points clearly.

After dilatation of the urethra a more normal curve and pattern is obtained. It is evident that the uroflowmeter may be useful, therefore, in indicating whether or not a dilatation is indicated in a stricture of the urethra (*Fig.* 376).

Obstruction at or near the Bladder Neck.—In this type of obstruction a completely different recording is obtained.

1. There is no initial rise, no plateau, and no terminal decline.

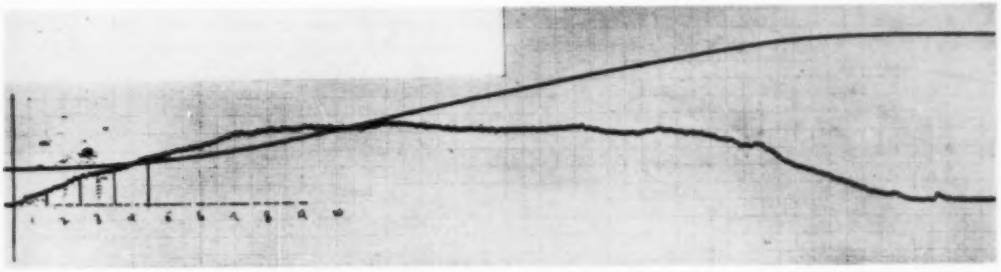

FIG. 373.—Normal micturition. Bladder volume 310 ml.

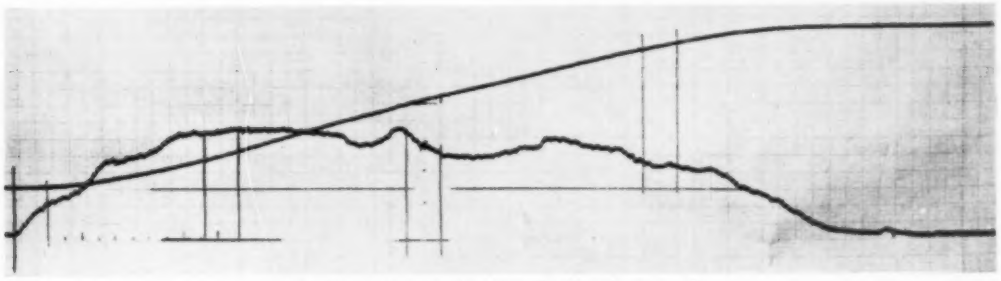

FIG. 374.—Normal micturition. Bladder volume 310 ml.

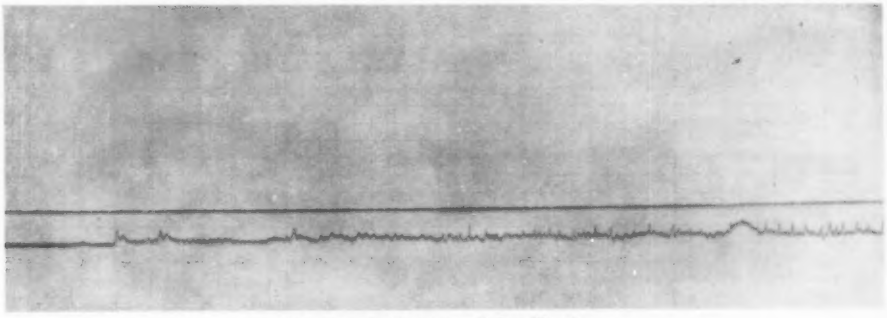

FIG. 375.—Penile stricture, before dilatation.

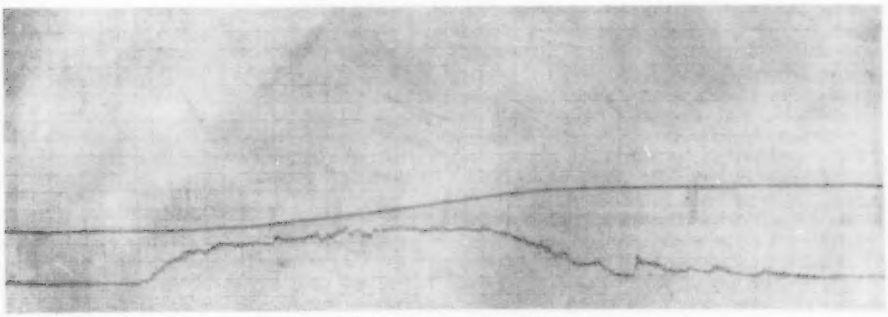

FIG. 376.—Penile stricture, after dilatation.

2. The lower line tends to be almost straight.
3. Oscillations are considerably diminished, showing that the rate of flow is more constant.

obtained by an obstruction at the bladder neck. In this case there are in fact two reservoirs, i.e., the bladder, and a second between the two strictures.

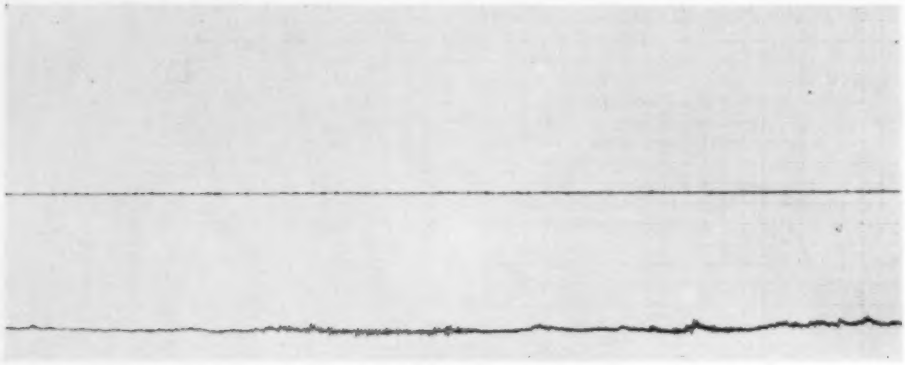

FIG. 377.—Prostatic obstruction, before operation.

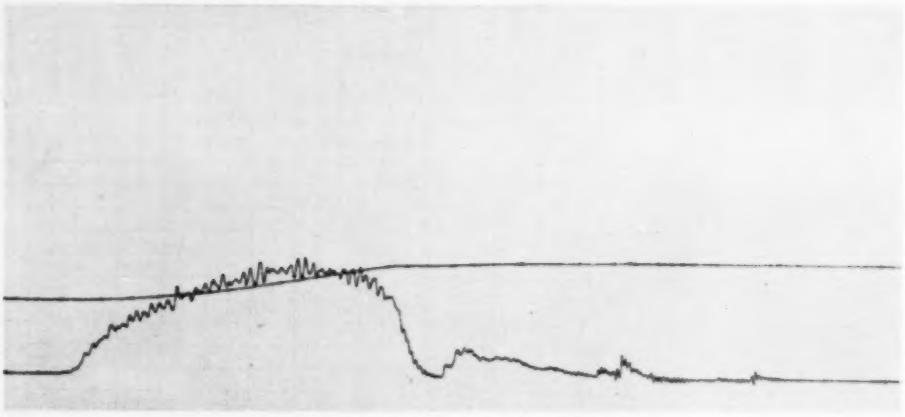

FIG. 378.—Prostatic obstruction, after prostatectomy.

Figs. 377, 378 show the tracings obtained before and after operation for prostatic obstruction.

DISCUSSION

Interpretation of the recordings obtained involves the physical principle that elastic tubes tend to damp down oscillations. For example, the pumping action of the heart is converted to an even flow by the time the blood reaches the capillaries, having in the meantime traversed the arterioles. An example in reverse is the water-hammer pulse.

We have observed that the nearer the stricture is to the external urinary meatus the more normal is the recording and the greater the variation of the rate of flow. On the other hand, if the obstruction is at the bladder neck, there is the whole length of the urethra to damp down the oscillations. As a corollary to this, it is obvious that in a patient with two strictures the proximal one will damp down the excessive oscillations and give a reading not dissimilar to that

Figs. 379, 380 show tracings of a double urethral stricture before and after dilatation. It is therefore not possible to distinguish an obstruction at the bladder neck from multiple urethral strictures by means of a recording.

We have attempted to correlate the uroflowmeter recordings with micturating cysto-urethrograms and have been impressed by the way in which the cysto-urethrogram confirmed the newer method. Similarly, clinical findings usually correspond with the uroflowmeter recordings which were always carried out before any other investigations had been performed.

CONCLUSIONS

Although we are fully aware that the value of the uroflowmeter may be limited, we have ventured to record our early impressions in the hope that it may prove of some value under certain circumstances. For example, it appears possible to indicate that, in a

given case of obstruction, the hold-up is situated either at the bladder neck or in the anterior urethra. Similarly, the type of recording obtained may indicate the necessity of redilatation in stricture of the urethra. Micturition is a complex mechanism and any method available for its further study deserves a full trial.

We are continuing our studies, using the uroflowmeter for the investigation of the neurogenic bladder.

We must record our thanks to the Research Committee of the Liverpool Board of Governors, under whose auspices this investigation has been carried out. We are also grateful to Professor Gregory, and members of the Department of Physiology of the University of Liverpool, for constant help and advice.

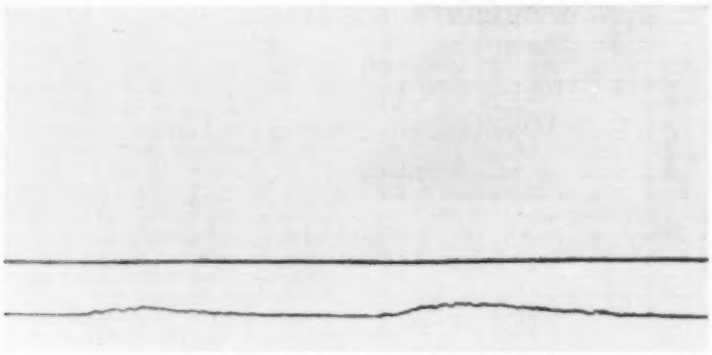

FIG. 379.—Double urethral stricture, before dilatation.

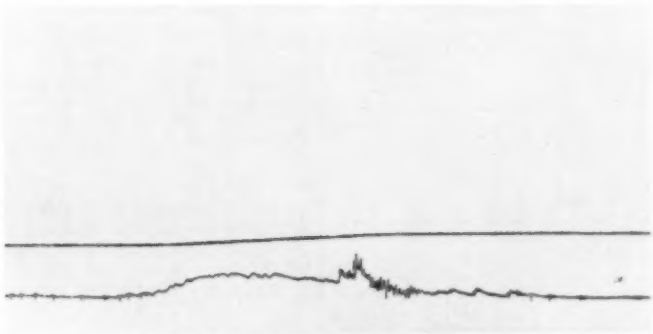

FIG. 380.—Double urethral stricture, after dilatation.

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SHORT NOTES OF RARE OR OBSCURE CASES

OBSTRUCTION OF THE BOWEL FOLLOWING PARTIAL GASTRECTOMY

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INTESTINAL obstruction due to herniation of bowel between an antecolic loop of jejunum and the transverse colon after a partial gastrectomy was first described by Stammers in 1952. Since then further reports have appeared (Oldham, 1953; West, 1953; Stammers, 1954; Knight and Bolt, 1959), and this type of obstruction must now rank as an accepted complication of the antecolic variation of the Polya-type gastrectomy. Stammers (1954) quotes a single case where such an obstruction occurred after a retrocolic procedure, the loop of obstructed bowel passing between the gastrojejunal anastomosis and the posterior parietal peritoneum. This was first described by Peterson in 1900.

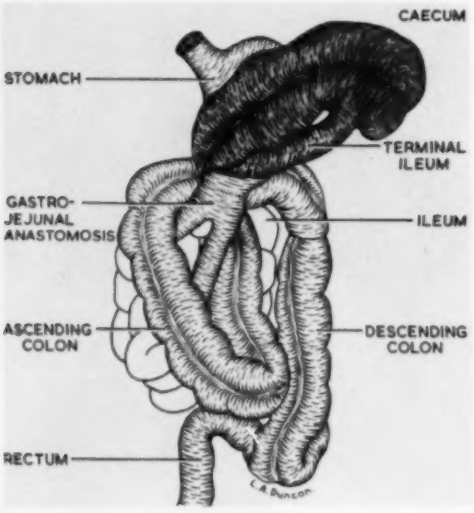

FIG. 381.

It is usually small bowel which passes through the surgically created hiatus, but on one occasion greater omentum has been recorded (Stammers, 1954). Entry to this hiatus may be either from the right side of the anastomosis or from the left side. Knight and Bolt (1959) report a case where almost all the small bowel was involved in such a herniation some 6½ years after the gastrectomy.

The case described below is unusual because cæcum and distal ileum comprised the obstructed loop, which had passed from left to right behind the anastomosis.

CASE REPORT

The patient, a woman aged 39 years, was admitted to hospital as an emergency on July 13, 1960. Sixteen hours before admission she developed generalized dull abdominal pain. Six hours later this pain became severe and colicky in nature and vomiting commenced. Her bowels had moved shortly before the pain started, but no flatus had been passed since the onset of the severe pain.

The patient had undergone two previous operations. In May, 1958, a chronically inflamed gall-bladder containing stones had been removed and at the same operation an appendicectomy was performed. On April 22, 1959, a Polya gastrectomy with an antecolic anastomosis was carried out for a chronic duodenal ulcer. She had been in good health since this second procedure.

ON EXAMINATION.—She was moderately shocked, with a pulse-rate of 90, and pale, cold extremities. Initial blood-pressure was 110/80 (her normal blood-pressure was 140/80), but this fell just before transfusion was commenced. The abdomen was distended centrally and in the left iliac fossa. She was extremely tender over the distended area, and her bowel-sounds were obstructive in nature, particularly during spasms of pain.

Plain radiography of the abdomen showed a distended loop of bowel extending from the left upper quadrant down to the pelvis. A diagnosis of small-bowel obstruction was made and laparotomy was performed.

OPERATION FINDINGS.—On opening the abdomen through a lower right paramedian incision, 2 pints of a whitish opalescent fluid were sucked off. The first bowel encountered was a grossly distended loop of large intestine passing down to the lower left abdomen. All the visible bowel had a peculiar bluish-white tinge, while the mesenteries and retroperitoneal tissues were very œdematous. After extending the incision, an enormously dilated viscus was found lying above and anterior to the gastric remnant. Only after evisceration of the small bowel was this distended organ identified as a very mobile cæcum and ileum which had passed up behind the gastrojejunal anastomosis from left to right and had there undergone further torsion, ultimately coming to lie under the left cupola of the diaphragm. The portion of bowel first seen was a large loop of ascending colon which dipped down from the orifice of the hernial ring into the left iliac fossa (*Fig.* 381). The obstructed loop was reduced relatively easily and the bowel proved to be viable. However, on returning the cæcum to the right iliac fossa, such mobility and redundancy of the right colon were present as to make recurrent volvulus very likely. Consequently, an exteriorization of the right colon and part of the right transverse colon was performed, leaving a double-barrelled colostomy with clamps in situ.

The patient made a good post-operative recovery and some three weeks later the colostomy was closed. She was discharged in good health three weeks after this second procedure, a mild fæcal leak from the colostomy closure having sealed off. She remains well ten months later.

COMMENT

In this case volvulus of the cæcum was complicated by passing behind a gastrojejunal anastomosis and undergoing further torsion. It is often recommended that the retrostomal space be closed by suitably placed sutures, but, as Tanner (1959)

remarks, this is almost impossible in the antecolic operation owing to the size and complexity of the space. The same writer draws the transverse colon to the right at the end of a gastrectomy, so that a short afferent loop comes to lie under the high and well-supported splenic end of the transverse colon. Undoubtedly this had been done in the present case, and the resultant mobility of the right transverse colon together with the free ascending colon and cæcum no doubt contributed to the volvulus. It was this enormous quantity of redundant colon that made an exteriorization resection advisable. One wonders whether the appendicectomy carried out through the Kocher incision at the time of cholecystectomy had played a part in leaving the cæcum mobile. This type of bowel obstruction is rare after a retrocolic procedure, and the recognition of this complication may well add merit to the retrocolic anastomosis as compared with the antecolic variation. The chylous effusion observed, combined with the milky appearance of the bowel and mesentery, suggests that chylous peritonitis accompanied this volvulus. The presence of free chyle has been reported by Hoffman (1954) in cases of obstruction involving the mesentery of the small bowel.

SUMMARY

A case is described of retrostomal space obstruction following partial gastrectomy. Abnormal fixation of the right colon caused the obstruction to consist of cæcum as well as ileum.

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TRAUMATIC ANEURYSM OF THE SUPERFICIAL TEMPORAL ARTERY

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"*The temporal artery, from its exposed situation, is liable to be wounded.*"—James Syme.

TRAUMATIC arterial aneurysm is not common in civil practice and, although the superficial temporal artery, and particularly its anterior branches, lie in a vulnerable position, this lesion has not been frequently described in this country. Only 4 cases could be traced as occurring in this century (Edington, 1903; Whiteford, 1915; Smith, 1949; and Tippitt, 1932). Two further cases are described here; in the first there was one aneurysm and in the other two aneurysms.

CASE REPORTS

Case 1 (Jan. 15, 1957).—Male, aged 35 years, a ship repairer. Six months previously, whilst at work, he had suddenly turned his head in response to a call, striking the left temporal region against an iron girder. This was followed rapidly by bruising and swelling in that region, which slowly disappeared over the course of a couple of weeks, leaving a small swelling above the lateral end of the left eyebrow. It throbbed at times but caused no pain and it had not increased in size.

ON EXAMINATION.—A fit, strong-looking man. No abnormalities in heart, R.S., or C.N.S. Pulse, regular; B.P. 120/80. There was a pulsatile swelling 1·5 cm. × 1·0 cm. situated 2 cm. above the lateral end of the left eyebrow. The overlying skin was normal in colour and there was no evidence of a previous wound. A systolic thrill was palpable and this disappeared on compression of the left superficial temporal artery in the pre-auricular region.

AT OPERATION (Jan. 18).—A transverse incision was made over the swelling. It was found to be a fusiform aneurysm, 0·5 cm. × 1·0 cm., of a small branch of the superficial temporal artery. It was excised and the ends of the artery ligated.

HISTOLOGICAL REPORT.—A false arterial aneurysm almost entirely occluded by a central mass of organizing thrombus surrounded by fibrous tissue. The latter shows numerous vascular channels in places.

FIG. 382.—*Case* 2.—Two aneurysms of the anterior branches of the right superficial temporal artery.

Case 2 (Oct. 21, 1957).—Male, aged 34 years, a bricklayer.

Four months previously this patient was struck on the right side of his forehead by a baseball. There was marked

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bruising of his forehead and around his right eye. This slowly disappeared, leaving two nodules on his forehead. He had had no pain or throbbing and attended only because the lumps persisted; they had not increased in size.

On Examination.—A very fit, strong man. There were no abnormalities in the heart, R.S., or C.N.S. Pulse was regular, 60/min.; B.P. 120/90. There were two small

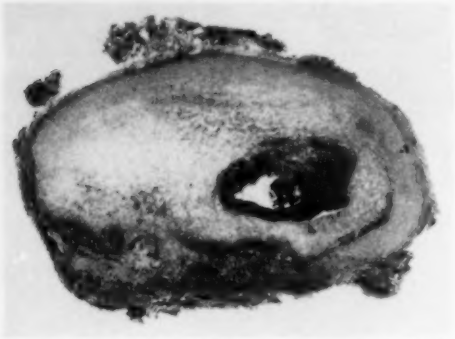

Fig. 383.—*Case* 2.—False aneurysm almost completely filled with recent organizing thrombus superimposed on old thrombosis. Hæmatoxylin and eosin. (× 8·5.)

lumps, each about 1 cm. in diameter situated above the lateral end of the right eyebrow (*Fig.* 382). A systolic thrill was barely palpable over one of these swellings. Pulsation was not definitely identified.

At Operation (Oct. 21).—Transverse incisions over swellings. Two small aneurysms, each about 1 cm. in diameter, were present and excised.

Histological Report.—A false arterial aneurysm which is almost completely filled with recent organizing thrombus superimposed on old thrombosis (*Fig.* 383). This applied to both aneurysms removed.

DISCUSSION

Thomas Bartholin first described this lesion in 1740 according to De Santi (Brown and Mehnert, 1942). In that case a child was struck on the head with a stick by its mother, whereupon bruising, swelling, and an aneurysm followed. Of 108 cases of aneurysm of this artery admirably reviewed by Winslow and Edwards (1934), 79 were arterial and due to trauma; 33 of these were associated with closed injury. In the rest there was a wound or the nature of the injury was not established. Included in this series was a number of arteriovenous aneurysms.

The type of injury has been varied. In Winslow and Edwards's (1934) review it is interesting to note that most of the cases occurred during the nineteenth century and then mainly during the early part of that period. At that time temporal arteriotomy was popular in the treatment of diseases such as apoplexy and epilepsy and duelling was a method of settling differences of opinion. Other causes recorded have been blows with sticks and balls, wounds from bullets (Whiteford, 1915), grenades and glass (Edington, 1903), pecks from hens, in motoring accidents, by the head striking the steering wheel (Pescovitz and Maloof, 1954) or the roadway in a fall from a motor-cycle (Tippitt, 1932), and the head striking a hard fixed object such as a girder (Smith, 1949). More recently cases have been described which were caused by a blow with an ice hockey 'puck' (Campbell, Fournier, and Hill, 1959). These authors have coined the term 'puck aneurysm'.

Syme (1863), despite his statement (*see above*), in his later years could recall having seen only 2 cases of aneurysm of this artery. Since 1934 (Winslow and Edwards) I have been able to trace only 2 further cases in the British literature (Smith, 1949; Tippitt, 1932) and 4 cases in the North American literature (Campbell, Fournier, and Hill, 1959; Pescovitz and Maloof, 1954; and Jahnke, Hughes, and Campbell, 1954). At the Walter Reed Army Hospital, where 182 traumatic aneurysms were treated from the Korean War, 3 involved the superficial temporal artery; 2 were arteriovenous, only 1 being purely arterial (Jahnke, Hughes, and Campbell, 1954).

Because of the position of this artery and the violent forces to which the head may be exposed in this modern world it is very surprising that this lesion is not more often observed. Is it that it passes unrecognized? The bruising, if it occurs, is considered to be a 'black eye' and the residual nodule regarded as some simple lesion such as a sebaceous cyst. Pulsation and thrill may be difficult to perceive as the sac may be almost completely occluded by thrombus in varying stages of organization (*Fig.* 383). Throbbing is sometimes present, as in *Case* 1, but *Case* 2 only attended because of the persistence of the swellings. Spontaneous cure is a possibility, though no really authenticated case has been described.

In the 2 cases of aneurysm described one was due to the head striking a hard fixed object, a girder, and in the other the head was struck by a moving object of high velocity. In both instances severe, localized, closed trauma occurred, causing rupture or partial rupture of the wall of the artery with the development of a small false aneurysm. In these instances it would appear that the arteries concerned were impacted against the anterior portion of that bony ridge termed the 'superficial temporal line'.

My thanks are due to Mr. J. S. H. Wade and Mr. H. O. Jones for allowing me to publish their cases; to Professor Lambert C. Rogers for his helpful advice; to Dr. F. Storring for the histology reports; to Mr. L. T. S. Williams and Mr. R. Marshall for the photographs.

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RUPTURE OF THE TESTIS

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INTRODUCTION

RUPTURE of the testis is a rare event. This paper presents an account of 2 patients with this injury whom we have recently treated, reviews previously reported examples of the condition, and discusses its pathology, diagnosis, treatment, and prognosis.

CASE REPORTS

Case 1.—R. H., an undergraduate aged 21 years (185995), was admitted to the Radcliffe Infirmary two hours after having been kicked in the groin while playing rugby.

On examination, there was a swelling in the right side of the scrotum which was extremely tender. The body of the testis was not palpable; there was no local bruising of the scrotal skin. The patient was in great pain but not shocked.

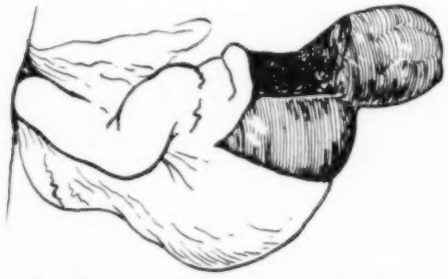

FIG. 384.—*Case* 1. Drawing of the ruptured testis as seen at operation showing protrusion of testicular tissue through the tear in the tunica albuginea.

Operation was carried out soon after admission (J. D. A.). The scrotum was incised and a hæmatocele under tension was evacuated. There was a ragged tear in the tunica albuginea with protrusion of the testicular tissue (*Fig.* 384). The testicular tissue was replaced partially with lightly tied catgut sutures and this raw area was covered with the tunica vaginalis. The wound was closed with drainage. Recovery was uneventful and when seen six months after this accident both testes were of equal size.

Case 2.—A. W., a factory worker aged 19 years (119682), was kicked in the groin while playing football. He felt nauseated and noticed some swelling of the scrotum. The pain increased in severity during the night following injury and when seen the following day the side of the scrotum was bruised, swollen, and tender.

At operation the same day (H. E.) a tense hæmatocele was evacuated through an incision in the right side of the scrotum. The lower pole of the testis was bruised and bore a 3-mm. tear in the tunica albuginea (*Fig.* 385).

The swollen testis was decompressed by a longitudinal incision in the tunica and the wound closed with drainage. Two years after the operation the right testis was normal in size.

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DISCUSSION

Cotton first reported a case of rupture of the testis in 1906. Since then Golji and Jaffar (1957) have collected 20 cases and added 1 example of their own. Schneiderman (1957) published a further 2 cases of traumatic rupture of the testis and Cassie (1956) recorded rupture of a seminoma produced by a trivial injury.

Type of Injury.—Rupture of the testis always follows direct violence. The testis is contained within a tough inelastic capsule, the tunica albuginea, and this can only be ruptured by a sudden compressing force. This is supported by considering the type of injury which produced a testicular tear in 24 recorded cases where details of the accident are given. The injury to a seminoma described by Cassie (1956) is excluded.

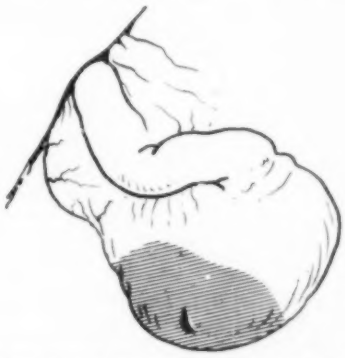

FIG. 385.—*Case* 2. Drawing of the testis as seen at operation showing the small tear in its lower pole and the surrounding area of bruising.

Causes	*Cases*	
Direct Violence	24	
Sporting Injuries	14	
	Football	12
	Hockey	1
	Baseball	1
Astride Injuries	4	
Traffic Accidents	2	
Miscellaneous	4	(Direct blow from belt buckle, sledge hammer, iron bar, and branch of tree)

The mobility of the testis within the scrotum accounts for the surprising rarity of injury to this organ. Because of this mobility, rupture will not occur unless it is trapped against firm resistance. McCrea (1951) suggested that this may be the thigh, but almost certainly rupture only occurs when the testis is forced against the bony resistance of the pubic symphysis or pubic rami. Contrary to what one might expect, none of the reported cases concerns injury to an undescended or ectopic testis lying fixed in the groin.

Pathology.—The macroscopic pathology of the injury seen at operation is as follows:—

1. *Bruising of the Scrotal Skin.*—This may be extensive or completely absent as in the first of our 2 cases.

2. *Hæmatocele.*—This is tense and usually sudden in onset following the injury.

3. *Rupture of the Tunica Albuginea.*—This is usually transverse and, through it, bruised and swollen testicular tissue may pout, prolapse, or even completely extrude. This latter finding occurred in the case reported by Long (1952).

Table I.—Management of Testicular Injury

Principle: The Testis must be Explored

Findings	Treatment
Hæmatocele	Evacuation of hæmatocele and drainage
Hæmatocele + Swollen and bruised testis	Evacuation of hæmatocele, decompression of testis by incision of tunica albuginea and drainage
Hæmatocele + Rupture of testis	
1. Rupture without protrusion of testicular tissue	Evacuation of hæmatocele, decompression of testis by incision of tunica albuginea and drainage
2. Rupture with partial protrusion of testicular tissue	Evacuation of hæmatocele, cover raw testicular tissue with tunica vaginalis, and drainage
3. Complete pulping or avulsion of the testis	Orchidectomy

Fig. 384 shows protrusion of the testicular tissue following rupture of the tunica albuginea, as seen in the first of our 2 cases.

Diagnosis.—Of course, there is always a history of injury followed by pain of varying severity. If the pain is severe there may be nausea, vomiting, and shock.

Inspection of the scrotum may or may not reveal bruising or abrasion of the skin. Swelling of the scrotum is usual, although this was absent in one of Sejournet's (1944) cases. The common pre-operative diagnosis is hæmatocele or torsion, and, in fact, until the scrotum is explored it is impossible to tell whether or not there is an underlying testicular rupture. Occasionally the patient presents some days or even weeks after injury, and then local heat and redness of the scrotum, together with swelling and pyrexia, suggest an acute epididymo-orchitis.

Management.—In 1895 Reclus warned that repair of an injured testis would inevitably lead to atrophy, and advised orchidectomy. However, survival of the testis without diminution in size occurred in both our cases as well as those reported by Campbell (1937), Dundon (1952), and Schneiderman (1957)—their patients having been followed up for an interval varying from seven months to seven years after the injury.

There seems little doubt that closed injuries of the testicle, unless trivial, should be explored. A scrotal incision gives direct access, and, provided the skin is meticulously prepared, is not liable to become infected. The tunica vaginalis is opened, the blood-clot which is normally under tension is evacuated, and the testis is examined. If this organ is undamaged, nothing more is required than suture of the skin incision, leaving a small corrugated drain into the tunica vaginalis. If the testis is swollen, bruised, and under tension, the tunica albuginea must be incised to decompress the organ and prevent pressure necrosis. If the tunica is ruptured hæmostasis is secured, the raw surface of the testis may then be covered by adjacent tunica vaginalis, and the wound closed with a small corrugated drain. If the testis is hopelessly pulped and completely extruded from the tunica albuginea, orchidectomy is performed. This management is shown schematically in *Table I.*

Failure to explore a hæmatocele may lead to one of several consequences. There is no doubt that minor hæmatoceles will absorb spontaneously and this occurred in 3 cases of such injury admitted to the Radcliffe Infirmary over the past twenty years. A large hæmatocele, if left alone, may eventually resolve, but only after weeks of discomfort. During this time there is always the risk of secondary infection or pressure atrophy of the testis, both preventable by evacuation of the hæmatocele. The main indication for operation, however, is that a simple hæmatocele cannot be differentiated from one complicating a rupture of the testis.

The majority of recorded cases were not explored for days, weeks, or even months after the injury, during which time the scrotum became gradually more distended and painful. Exploration would then be undertaken only to find a disorganized testis which had to be removed.

Early exploration of a hæmatocele is therefore advised to exclude an underlying testicular injury and thus also prevent the complications of ischæmic atrophy and secondary infection.

SUMMARY

1. Two cases are described of rupture of the testis treated by early operation.

2. The management of testicular injuries is discussed.

3. Early exploration is advised in all cases to prevent the complications of ischæmic atrophy and secondary infection.

Acknowledgements.—We are indebted to the late Mr. D. A. Abernethy and Mr. A. Elliot Smith for permission to publish these case reports of patients admitted under their care. We also wish to thank Professor T. Pomfret Kilner for his helpful advice and criticism. We would like to thank Miss McLarty for drawing *Figs.* 384, 385.

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REVIEWS AND NOTICES OF BOOKS

A Symposium on *p*H and Blood Gas Measurement. Edited by RONALD F. WOOLMER, V.R.D., B.A., B.M., F.F.A.R.C.S., Professor of Anæsthetics in the Royal College of Surgeons of England. 8½ × 5½ in. Pp. 210 + viii, illustrated. 1959. London: J. & A. Churchill Ltd. 30s.

No symposium can hope to be fully comprehensive. In this case the chairman has concentrated on the growing points of the subject and on much-needed reviews of the older techniques. Much of the material in the symposium is not to be found in text-books or in publications. Although it was primarily a meeting of anæsthetists, the symposium included surgeons, physicians, physiologists, physicists, and biochemists, including many of the more important contributors to recent work in the field. While much of the book is of interest only to those concerned with the esoterics of blood gas measurement, a great deal finds direct application in clinical surgery. It is stressed that the measurement of *p*H and blood gas tensions is in a similar position to the determination of serum-electrolytes a decade ago. A rare and difficult measurement was about to become commonplace and take its place alongside the blood-urea glucose as an indispensable aid to diagnosis and treatment.

The first part is concerned with the measurement of *p*H and the CO_2 tension by the Henderson–Hasselbalch equation. This section is of particular value to those responsible for the establishment of these techniques where facilities have not previously existed. Much of the information cannot easily be found elsewhere.

The second part deals with the interpolation and rebreathing methods for determination of CO_2 tension. These techniques are now coming into general clinical use and are markedly simpler than earlier methods.

The third part includes the only reference to the measurement of blood oxygen—the polarographic technique for determination of oxygen tension. There is also a description of the CO_2 sensitive electrode—probably the method of the immediate future for the measurement of blood CO_2 tension. Finally, there is an appraisal of the value of the end-tidal (alveolar) sample and a paper on the clinical significance of *p*H and blood gas values.

For those concerned directly with the measurement or interpretation of *p*H and blood gas levels, this symposium is one of the most valuable publications to have appeared. For the general surgeon its value is less immediate, but it serves as an excellent preview of a subject whose importance is rapidly growing.

Einfachheit und Sicherheit bei Magen- und Darmoperationen. By HEINRICH WESTHUES. 11 × 7½ in. Pp. 75 + viii, with 87 illustrations. 1961. Stuttgart: Georg Thieme. DM.29.

THIS book, based on the author's 35 years' experience of gastro-intestinal surgery, seeks in the first place to solve the problem of aseptic intestinal anastomosis which has intrigued so many surgeons over the past eighty years. To this end the author uses a special heavy 'steam-hot' clamp to necroze and sterilize an area of gut, which is to form the centre of the stoma, and sutures around this area by a seromuscular row of interrupted sutures alone. The technique is claimed to eliminate the need for much of the present-day bowel sterilizing, and to make leakages and stenosis of the anastomosis almost impossible.

The salient technique is only one of the interesting features in this instructive and fascinating volume which ranges over the whole field of gastro-intestinal surgery, including the intriguing problems associated with carcinoma of the rectum and conservative resection with preservation of anal function: the author favours such conservatism much more than the majority of surgeons in this country, and bases his views on an impressive study of pathological anatomy.

The illustrations and production are well worthy of the long tradition of Georg Thieme and add much to a volume which any surgeon who reads German will find interesting and stimulating. The bibliography is international and good.

Die Präoperative Röntgenbestrahlung des Mammakarzinoms. By E. MUNTEAN. 9½ × 6½ in. Pp. 108. Illustrated. 1961. Stuttgart: Georg Thieme. DM. 17.50.

THE author discusses the various combinations of radiotherapy with surgery in the treatment of breast cancer. The subject is dealt with in detail and a good case is put forward for pre-operative therapy whenever this is practicable. Doctor Muntean advocates a moderate dosage (1000–2000 r), followed by early surgery (9–14 days later). The histological assessment of the excised tumour helps in deciding on further post-operative therapy. Co-operation between surgeon and therapist is essential and each case must be treated on its own merits.

The chapters devoted to the advantages and disadvantages of pre-operative radiotherapy and to the techniques are particularly well written. There is an extensive and up-to-date bibliography which ought to be invaluable to anyone studying this subject; every authority, either modern or old, who has written on the combined treatment of breast cancer is quoted at some stage in this monograph.

The Medical Annual 1961. Seventy-ninth issue. Edited by R. BODLEY SCOTT and R. MILNES WALKER. 8½ × 5½ in. Pp. 610, with 33 illustrations and 75 plates. 1961. Bristol: John Wright & Sons Ltd. 42s.

THE *Medical Annual* 1961 lives up to its high reputation and gives the medical profession an excellent survey of what is new and established in the diagnosis and treatment of current conditions and diseases.

For the surgeon, Professor Milnes Walker and his collaborators have given an admirable précis of all that is new in every branch of Surgery.

It is difficult to read all the surgical journals from every part of the world, but the *Medical Annual* tries its best to keep one up to date in this respect.

The special articles are of great interest, especially those on the management of paraplegia by Guttmann, and microsurgery by Angell James.

The *Medical Annual* is just one of the books which must be found on the desk of every keen clinician, whether he be a practitioner or specialist.

Cerebral Palsy in Childhood and Adolescence. By J. L. HENDERSON, M.D., F.R.C.P.E., Professor of Child Health, University of St. Andrews; Consultant Pædiatrician, Eastern Regional Hospital Board, Scotland. 8½ × 5½ in. Pp. 404. 1961. Edinburgh: E. & S. Livingstone Ltd. 35s.

THIS monograph may be said to be a medical, psychological, and social study of cerebral palsy.

The book is divided into six parts and the author has managed to engage some ten contributors each of whom has given an able presentation of some aspect of cerebral palsy.

Part 1 is a survey of cerebral palsy in the Eastern Region of Scotland. The incidence of the disease, its prevalence, and the social class involved are well portrayed.

Part 2 deals with the medical, neurological, and orthopædic aspects of the condition.

Part 3 is concerned with the associated disabilities; the ophthalmological aspects, deafness, dental features, and blood-group features.

Part 4 contains an interesting account of the psychological and social aspects of the disease.

Part 5 is devoted to some aetiological considerations; while

Part 6 contains the conclusions and recommendations for the care of patients with cerebral palsy in the Eastern Region of Scotland.

A very informative monograph on an interesting condition.

Report No. 2. VII^e Congrès International d'Oto-Rhino-Laryngologie. Vol. 9: Cancer of the Larynx. By J. LEROUX-ROBERT. 9¼ × 6⅝ in. Pp. 323 + xii, with 18 illustrations. 1961. Basel: S. Karger. S.Fr.36.

THE communications presented at this Congress provide a detailed and wide review of the subject from various aspects. Not only are the opinions of the invited authorities presented, but there are also included the views of many other laryngologists from many countries.

Although there is of necessity some divergence of opinion, yet the study of this volume allows balanced conclusions to be attained as to the appropriate treatment, not only of cancer affecting the interior of the larynx (i.e., the vocal folds and subglottic region), but also of growths involving the aperture with its aryteno-epiglottic folds and also the post-cricoid region.

In general, surgery is preferred to irradiation for extensive growths arising on or spreading to the supraglottic structures and also for neoplasms on the lateral or posterior walls of the larynx, usually arising from the pyriform fossa or near the mouth of the œsophagus.

Surgery is also preferred for the treatment of metastases in lymphatic glands and is recommended as a prophylactic measure without waiting for palpable nodes; this was emphasized in the communication of the late Professor Pietrantoni of Milan, presented by D. Agazzi. Waiting for the appearance of palpable metastases meant losing 30 per cent of possible cures obtainable by prophylactic treatment.

Professor Alanso, of Montevideo, advocates his well-known horizontal supraglottic partial laryngectomy, with preservation of the vocal folds; the excellent results of this procedure, with preservation of voice, were acclaimed by other speakers and are known to the present reviewer by personal observation.

Leroux-Robert, of Paris, repeated the generally accepted decision to remove lymph-nodes in cases of supraglotic neoplasm, while omitting this procedure in glottic and subglottic cases.

He referred to the saving of time when cordal growths are treated by surgery rather than by radiotherapy, an important consideration in workers for whom a perfect voice is not essential.

The importance of the pre-epiglottic space was emphasized in the communication of the late C. L. Jackson, of Philadelphia, presented by his collaborator, Dr. Norris. They had almost entirely discarded narrow-field laryngectomy and on the contrary considered radical neck dissection to be essential in cases other than those in which the vocal folds or subglottic region alone are involved.

Casadesus, of Barcelona, found total laryngectomy to be indicated for most extensive growths, sometimes combined with partial pharyngectomy.

Radical surgery of the hypopharynx was discussed by Ormerod, of London; he referred to the swing from surgery to irradiation and back to surgery in the last forty years.

Van H. Leicher, of Mainz, discussed the relative merits of teleradiation with radium or cobalt as compared with contact irradiation, and usually preferred the former.

In general, the views expressed at this Congress agreed with the practice of surgeons in this country.

Home Treatment in Injury and Osteo-arthritis. By W. E. TUCKER, C.V.O., M.B.E., T.D., M.A., F.R.C.S., Consulting Orthopædic Surgeon, Royal London Homœpathic Hospital. 8¾ × 5½ in. Pp. 80, with 17 illustrations. 1961. Edinburgh: E. & S. Livingstone Ltd. 10s. 6d.

THIS small practical monograph should prove of value to the general practitioner who so often finds many patients on his hands with backache and other ailments.

The home exercises advocated by Mr. Tucker should prove of real value to patients with osteo-arthritis. The line drawings which illustrate these exercises are excellent.

Logan Turner's Diseases of the Nose, Throat, and Ear. Sixth Edition. Edited by JOHN P. STEWART. 8¾ × 5⅝ in. Pp. 508, with 249 illustrations and 14 coloured plates. 1961. Bristol: John Wright & Sons Ltd. 52s. 6d.

THIS well-known book first saw the light of day in 1924 and now the sixth edition has appeared. It is the product of the Edinburgh School and through the years different surgeons have been called upon to supply new editions. To-day John P. Stewart is the Editor, and he is assisted by R. S. Lumsden. Malcolm Farquharson has taken over the section on "Peroral Endoscopy", and G. D. MacDowall that on "Affections of the Paranasal Sinuses".

One of the features of this book is the large number of excellent illustrations, and there are 14 beautiful coloured plates.

There can be no doubt that the first Editor of this book would be most gratified with the new sixth edition.

This is a very practical book and the list of prescriptions—which has proved useful to many readers—is now fully revised and is included at the end of the book.

An excellent production with which the publishers have every cause to be more than satisfied.

The price of the book is most moderate.

Clinical Radiology of the Œsophagus. By MARCEL BROMBART. Translated by SHEILA KENNY, M.A., M.D. (Dubl.). 9¾ × 7¼ in. Pp. 404, with 337 illustrations. 1961. Bristol: John Wright & Sons Ltd. 84s.

THIS is a most comprehensive work which is profusely illustrated with excellent radiographs. The first chapter sets out the examination technique in detail—the contrast media, the containers and accessories, and a full explanation as to how the examination is carried out.

Succeeding chapters deal with anatomical features of the œsophagus and the physiological considerations. The radiological appearances of the individual segments of the tube in health and disease are discussed fully. Diverticula, ulcers, tumours, varices, and hiatus hernia are dealt with in a masterly way.

As a work of reference this book will remain outstanding for a long time. It will also appeal to all clinicians and gastro-enterologists, and, of course, to radiologists.

The author knows his subject from many years of practical experience and the text is well presented. There is a most exhaustive bibliography which the reader will find most useful.

We would like to congratulate Dr. Sheila Kenny on her very lucid translation of this work.

The whole book is beautifully produced in the well-known John Wright style.

Surgery of Trauma in the Tropics. By R. L. BATTEN. 8½ × 5½ in. Pp. 234 + viii, with 178 illustrations. 1961. London: Edward Arnold (Publishers) Ltd. 35s.

THIS small book should be of value to all who have to treat injuries in tropical districts where modern equipment is not to be found. The treatment of fractures and the application of plasters is very sound.

There is an excellent chapter on anæsthetics written by Dr. J. V. Farman.

Introduction to Anatomy, 1532. By DAVID EDWARDES. English translation by C. D. O'MALLEY and K. F. RUSSELL. 8½ × 5½ in. Pp. 64. 1961. London: Oxford University Press. 15s.

THIS small monograph will profit all medical men who are interested in the history of medicine during the Tudor period. It must be remembered that it was David Edwardes who first recorded the dissection of a human body in England in 1531. He also wrote the first book on anatomy in England.

We have in this monograph a facsimile reproduction with English translation and an introductory essay on anatomical studies in Tudor England.

The Shaking Palsy (Parkinson's Disease). A Symposium. By HAROLD ELLIOT, M.D., and BLAINE NASHOLD, M.D. 10 × 6½ in. Pp. 160 + xxii. Illustrated. 1961. McGill University Press. $5.00.

THIS monograph contains the papers, and discussions which followed them, which were presented at a Symposium held at Queen Mary Veterans Hospital, Montreal, in October, 1959. The various papers cover every aspect of this curious disease, yet in spite of all this knowledge we are no nearer a permanent cure. There is no doubt that a great deal more work is needed in the field of research concerning Parkinson's disease.

Some facts are forthcoming and it is interesting to note that early depression in the Parkinson patient is probably a result of shunning social intercourse, and it should be combated.

Depressive symptoms of psychotic intensity can be helped by electric shock treatment. The use of chemopallidectomy gives some fair results but is by no means without risk.

An excellent bibliography is included in this symposium.

Psychotherapeutic Techniques in Medicine. By MICHAEL and ENID BALINT. 8½ × 5½ in. Pp. 236 + xvi. 1961. London: Tavistock Publications. 21s.

THIS book is rather outside the ambit of surgeons accustomed to having a pathological basis for the lesions with which they deal. Psychopathology appears to be just another term in psychiatry, lacking a concrete basis. While the behaviour of those quoted here as cases may make diverting reading, that discussion of their behaviour with them can alter both it and associated clinical features is a *post hoc ergo propter hoc* type of argument. The whole conception of the book is of a type "to arouse interest in its psychological origin rather than in a serious discussion of its merits".

Night Calls: A Study in General Practice. By MAX B. CLYNE. 8½ × 5½ in. Pp. 216 + xvi. 1961. London: Tavistock Publications. 21s.

THIS is a book on General Practice with plenty of clinical cases. The subject-matter is unique, for never before has so much been gained from pooled experience.

A team consisting of eight general practitioners and a psychiatrist studied the events that occur, overtly or convertly, in both the patient and the doctor, before, during, and following a night call.

The clinical material in this book is most interesting.

Theory of Shoulder Mechanism: Descriptive and Applied. By A. K. SAHA, B.Sc., M.B., B.S., F.R.C.S. (Eng.), F.R.C.S. (Edin.), M.Ch.Orth. (L'pool), Professor of Surgery. 9 × 6 in. Pp. 107 + xii, with 78 illustrations. 1961. Illinois: Charles C. Thomas (Oxford: Blackwell Scientific Publications Ltd.). 44s.

THIS monograph describes in detail the various operations that the author has devised for the treatment of recurrent dislocation of the shoulder; also the treatment of unimpacted abduction fractures of the neck of the humerus.

The chapter on rehabilitation of the paralysed shoulder is particularly good.

A History of Thoracic Surgery. By RICHARD H. MEADE, B.S., M.D. 9 × 5½ in. Pp. 933. Illustrated. 1961. Illinois: Charles C. Thomas (Oxford: Blackwell Scientific Publications Ltd.). £11.

THIS is an interesting and authoritative volume which deals in a comprehensive manner with the history of Thoracic Surgery. The early history is told in a fascinating way, and there is a very complete set of photographs of the men who have taken part in its evolution. No branch of Surgery has progressed so fast in the last two decades as that which concerns the contents of the thorax.

It is interesting to note that the author considers that Dr. I. W. Magill has played an important part in the surgery of the thorax, insomuch that he was such an important contributor to the development of intratracheal anæsthesia. Indeed, to-day, all thoracic surgeons will agree that modern anæsthesia has made their task possible.

From this large volume it can be seen that surgeons in Britain and America have played the major part in making thoracic surgery safe for the patient.

There is a special biographical section with very complete references, which will prove of real value to all who are interested in the surgery of the chest.

As a reference volume this work could not be bettered, and all medical libraries should contain a copy.

Research in Physiopathology as Basis of Guided Chemotherapy. By EMANUEL REVICI, M.D. 9½ × 6 in. Pp. 772 + xxvi, with 298 illustrations. 1961. London: D. van Nostrand Co. Inc. £9.

THIS large work contains an account of the author's researches into the problem of cancer. The approach to this problem from the chemico-pharmaceutical point of view is excellent, but the author's idea of biologically guided therapy will require further study.

The author's basic research may of course open up a new discipline in medicine.

Handbuch der Urologie. Edited by C. E. ACKEN, V. W. DIX, H. M. WEYRACH, and E. WILDBOLZ. In 13 volumes. 9½ × 6½ in. Vol. I: Operative Urology by W. BISCHOF, P. BISCHOFF, C. FRANKSSON, R. FREY, J. H. HARRISON, J. HELLSTROM, and W. TONNIS. Pp. 460 + xii, with 183 illustrations. 1961. Cloth. DM.176; by Subscription DM.140.80. Vol. III: Symptomatologie und Untersuchung von Blut, Harn und Genital Sekreten by KARAL HINSBERG, JOSEPH KIMMIG, JOHANNES MEYER-ROHN, R. H. O. B. ROBINSON, CARL SCHIRREN, and RENATE WEHRMANN. Pp. 337 + xiv, with 61 illustrations. 1960. DM.118. Berlin: Springer.

Volume III, the first to appear, deals with Symptomatology and Examination of the Blood, Urine, and Genital Secretions. The book opens with a section in English by Mr. R. H. O. B. Robinson, devoted to the significance of abnormalities of the urine in quantity and composition. It is truly said that examination of the urine forms the keystone of any investigation of the urinary tract, and the methods of ordinary clinical examination are given in

detail. The remaining sections which form the bulk of the volume are in German and deal in great detail with the methods of qualitative and quantitative estimation of the urine and blood. There is an excellent chapter on the bacteriology of the urine, enriched by many illustrations in colour. The section on the examination of the genital secretions covers microscopy and chemical, steroid, and hormone analysis. This volume forms a complete laboratory manual.

Volume I, the first volume on Operative Urology, covers anæsthesia, operations on the kidneys, plastic operations for hydronephrosis, adrenal surgery, and operations on the nervous system as it affects urology. The main section on the kidney is in English, written by Hellström and Franksson, and gives a comprehensive account of all renal operations except those covered by Bischoff's chapter on hydronephrosis. The methods of treating calculous disease are given in detail and are well illustrated. In the account of partial nephrectomy little mention is made of the segmental distribution of the renal artery as described by Graves, although his paper is quoted in the references. Hartwell Harrison gives a scholarly account of the surgery of the adrenals—a subject of increasing interest to urologists.

These two volumes maintain the high standard of production and completeness set by those already published.

Essentials of Pædiatric Surgery. By H. H. NIXON, M.A., F.R.C.S., and BARRY O'DONNELL, M.Ch., F.R.C.S., F.R.C.S.I. $8\frac{1}{2} \times 5\frac{1}{2}$ in. Pp. 315 + xii, with 65 illustrations. 1961. London: William Heinemann Ltd. 40s.

THIS small monograph contains much of interest to the senior registrar and the younger generation of surgeons who are interested in pædiatric surgery. The authors rightly stress the importance of accurate clinical diagnosis in children if surgical treatment is to be successful.

Postgraduates working for the Diploma of Child Health will find this book of real value.

Spatial Vector-Cardiography. By HUGON and ZOFRA KOWARZYKOWIE. $9\frac{1}{2} \times 6\frac{3}{4}$ in. Pp. 254, with 288 illustrations. 1961. Oxford: Pergamon Press Ltd. 80s.

THIS volume sets out very well the important work performed by some of the Polish experimentalists on two kinds of vector cardiograms—axonocardiograms and stereograms.

The book is divided into two parts: in the first part a general introduction to the subject is presented and some excellent axonocardiograms and stereocardiograms are reproduced. The second part has many excellent reproductions of comparative axonocardiograms and diagrams which make the book an important reference volume.

1960–1961 Series: Year Book of Orthopedics and Traumatic Surgery. By H. HERMAN YOUNG, M.D. $7\frac{1}{2} \times 5\frac{1}{2}$ in. Pp. 440, with 219 illustrations. Illinois: Year Book Medical Publishers Inc. 68s.

THIS Year Book contains an excellent review of the world's current orthopædic literature, and the author has managed to pick out the outstanding articles which he has considered worth including in this volume. This is no easy task when it is realized how vast the international orthopædic literature has become in recent years.

The numerous illustrations throughout the book are excellent and very clear indeed.

The chapter on Rheumatic Diseases is a large one, but it includes all the modern research results which have been accumulating over the past year.

The section on Plastic Surgery, which is edited by Neal Owens, is well presented and illustrated.

This is an up-to-date synopsis of modern orthopædic and traumatic surgery, and as such will prove useful, not only to the young orthopædic surgeon, but also to the many orthopædic registrars who want to keep abreast of modern surgical opinion.

Pseudarthrosis of the Long Bones. By OTTO VERBEEK, M.D., and C. P. DUBBLEMAN, M.D. $11\frac{1}{2} \times 8$ in. Pp. 235 + vii, with 616 illustrations. 1961. Amsterdam: North Holland Publishing Co. 90s.

THIS book sets out the various methods which can be used in the treatment of pseudarthrosis of the long bones. More than half the book is devoted to series of radiographs showing the progress in treatment following operation. The authors talk about 'young' pseudarthrosis and 'old' pseudarthrosis, but it is difficult to differentiate these.

It must be accepted that in delayed union healing of the fracture may still be expected, but in pseudarthrosis no union can be expected without a special intervention. There are many useful tables giving the average duration of healing of fractures resulting from accidents. The compound fractures of the tibia on the average take 4–6 months to heal.

It is revealed that fixed autogenous bone-grafts following freshening of the pseudarthrosis yield more reliable results than any other method.

A useful reference book.

La Maladie Ulcéreuse de L'Estomac. Vol. I. Edited by GUY ALBOT and FELIX POILLEUX. $9\frac{1}{2} \times 6$ in. Pp. 290, with 84 illustrations. 1961. Paris: Masson et Cie, Éditeurs. 42 NF.

THIS book, like its predecessors, is the outcome of the monthly conferences held at the Hôtel Dieu, in which a great number of French gastro-enterologists participate. It is a collection of short lectures rather than of original papers, though a considerable number of personal observations is included. The title 'peptic ulcer of the stomach' is somewhat misleading, as no full account of this entity is given. Instead, its general pathology is adequately covered in the first two sections: 'the experimental gastroduodenal ulcers' and 'gastroduodenal ulcer in man'. The latter contains a very useful article for the surgeon by Hollender, entitled 'the causes and prevention of anastomotic ulcers'.

A little farther in the book, in sections 5 and 6, two varieties of peptic ulcer are described in considerable detail: post-operative ulcers and ulcers of the second part of the duodenum.

As contrasted with peptic ulcers, the symptomatic ulcers (pharmacological, in cirrhoses, etc.) and simple ulcers are extensively discussed from every point of view, in sections 3 and 4. All contributions are written in the traditional clear French style, with pleasant subdivisions and illustrations of good quality. The book provides an important and valuable source of information for surgeons, gastro-enterologists, and radiologists.

Nouvelle Pratique Chirurgicale Illustrée. Part XVII. Edited by JEAN QUÉNU. $10\frac{1}{2} \times 6\frac{3}{4}$ in. Pp. 270, with 225 illustrations. 1961. Paris: G. Doin et Cie. 41 NF.

THE series, of which this volume is the latest number, sets out to demonstrate the surgical practice of the authors by describing various operations, each by a sequence of full-page line drawings.

Although for the surgical acolyte this method is no substitute for watching or assisting at operations, it should have at least two advantages: that operations can be studied in an orderly systematic manner and that variations of method and technique can be studied simultaneously. However, in this series, these advantages are disregarded, the first because there is no systematic arrangement of the operations described, and the second

because the illustrations are strictly confined to the single case selected.

The operations described in the volume under review are:—

1. Plurisegmental pulmonary resection with decortication, parietal pleurectomy, and osteoplastic thoracoplasty (Björk's operation) for pulmonary tuberculosis.
2. Operation for epigastric incisional hernia using nylon implant.
3. Choledochotomy, sphincterotomy, and cholecystectomy for gall-stones.
4. Operation for complicated extrasphincteric anal fistula.
5. Operation for phimosis in an adult.
6. Treatment of infected un-united fracture of the leg by external compression-fixation apparatus.

The drawings are excellent, but they are unnecessarily numerous. For example, there are 68 to illustrate the first operation. Such prolixity is not really helpful, and many could well be omitted or replaced by others showing variations of technique and method to deal with different circumstances.

Die Vaginale Radikaloperation. By HANS HÖGLER. 9½ × 6½ in. Pp. 131 + vii, with 47 illustrations. 1960. Wein-Innsbruck: Verlag Urban & Schwarzenberg. DM.60; ÖS.177.60.

WITH greater recourse to radical surgery in the treatment of cervical cancer in this and many other countries, it is a curious fact that Schauta's vaginal procedure, which is steadily gaining popularity elsewhere, is still totally out of favour in Britain. Weighty as the well-known theoretical objections to this operation may appear, recently reported results must speak for themselves.

This little monograph, which concludes with an impressive analysis of 254 cases of mainly Stage 1 and 2 cancers treated operatively, 80 per cent by the Schauta–Amreich technique, seeks to popularize radical vaginal hysterectomy by dealing with one of its pitfalls, location and safeguarding of the ureters.

Correlating endoscopic appearances with the topography of the ureters at operation, the author has shown that it is possible to predict the juxta-vesical course of the ureters at cystoscopy. In then describing fully his own method, a slightly modified version of the Schauta–Amreich operation, he guides the reader, with the aid of numerous clear illustrations, through the whole procedure. Those privileged to see Dr. Högler performing (as at the recent International Congress of Obstetrics and Gynæcology in Vienna) bear witness to the practical value of his method.

An English translation of this book might well boost radical vaginal hysterectomy in this context in Britain and the Commonwealth.

Tumours of the Œsophagus. Vol. 4. Edited by N. C. TANNER, M.D., F.R.C.S., and D. W. SMITHERS, M.D., F.R.C.P., F.F.R. 9½ × 6½ in. Pp. 352 + xi, with 165 illustrations. London: E. & S. Livingstone Ltd. 63s.

THIS is the fourth volume of the well-known series on Neoplastic Disease at various sites which have been published under the general editorship of Professor Smithers.

The present volume is the most comprehensive book which has so far been published on the subject of tumours of the œsophagus, and the combination of a surgeon with a radiotherapist is most happy. These two have co-opted a few experts who have written on special aspects of the problem of cancer of the œsophagus. This book is beautifully illustrated with over one hundred and sixty illustrations.

An excellent chapter on the mortality-rates from cancer of the œsophagus has been written by R. A. M. Case.

This book will be the reference volume on cancer of the œsophagus for many years to come.

Malformations of the Face. By D. GREER WALKER, M.A., M.D., B.Ch., M.Dent.Sc., F.D.S. R.C.S. 9½ × 6¾ in. Pp. 202 + xii, with 93 illustrations. 1961. London: E. & S. Livingstone Ltd. 37s. 6d.

THIS is a most useful book which will interest the anatomist, the embryologist, the geneticist, and, not last, the surgeon who has to undertake plastic repair of the face.

The author, with his matured clinical background, has written a fascinating book, in which he discusses the many and varied conditions, both congenital and acquired, which are met with in deformities of the face. The clinical photographs taken before and after treatment are excellent, and show the real importance of using photographs in the clinical records. Every variety of congenital defect of the face is well described and illustrated.

Professor Pomfret Kilner, who writes a foreword to this book, considers that a book of this nature has long been needed, and we wholeheartedly agree with him.

An excellent production.

Textbook of Genito-Urinary Surgery. Second Edition. Edited by H. P. WINSBURY-WHITE, M.B., Ch.B. (Edin.), F.R.C.S. (Edin.), F.R.C.S. (Eng.), and J. D. FERGUSON, M.A., M.D., F.R.C.S. 9½ × 6¾ in. Pp. 988 + xvi, with 472 illustrations. 1961. London: E. & S. Livingstone Ltd. £6 10s.

THE second edition of this British work on genito-urinary surgery has been entirely re-written, and J. D. Fergusson has joined Winsbury-White in editing this large and comprehensive volume. The editors have gathered around them some forty contributors and every aspect of genito-urinary surgery has been covered by an expert in his own subject. During the last decade great progress has been made in urology and this progress is reflected in this volume. It is a very representative work on urology as practised in the British Isles to-day. The book is well illustrated and the comprehensive index is accurate.

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The British Medical Dictionary. Edited by Sir ARTHUR SALUSBURY MACNALTY, K.C.B., M.A., M.D. (Oxon.), F.R.C.P., F.R.C.S. 11 × 8 in. Pp. 1680 + xix. 1961. London: Caxton Publishing Company Ltd. No price given.

AT last, an up-to-date comprehensive medical dictionary compiled by over one hundred British medical men and edited by that doyen of medical literature, Sir Arthur MacNalty. We have nothing but praise for this modern medical dictionary, for the Editor has gathered around him experts in every branch of medicine and surgery who have performed their individual tasks excellently.

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Traumatic Aplasia. By W. RITCHIE RUSSELL, C.B.E., M.D.(Edin.), D.Sc.(Oxon.), F.R.C.P.(Edin. & Lond.), Director, Department of Neurology, United Oxford Hospitals, and M. L. E. ESPIR, M.A., M.B., B.Chir., M.R.C.P. (Lond.), Consultant Neurologist to the Leicester Group of Hospitals. 9½ × 6¼ in. Pp. 180, with 139 illustrations. 1961. London: Oxford University Press. 38s.

THIS is one of the well-known Oxford Neurological Monographs. During and after the Second World War over 1100 cases of missile wounds of the brain were studied in detail from the Military Hospital for Head Injuries, Oxford, and these included over 250 patients rendered aphasic by the injury. An analysis of these cases is presented in this monograph.

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An Introduction to Clinical Electro-encephalography. By R. R. HUGHES. 8¼ × 7¼ in. Pp. 126, with 76 illustrations. 1961. Bristol: John Wright & Sons Ltd. 30s.

THE publication of this book will be welcomed by the medical profession as a whole, but it will be especially welcomed by clinicians.

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The author includes records on about 15,000 cases at the United Liverpool Hospitals, E.E.G. Department, at the Royal Southern Hospital, Liverpool, and also about 4000 cases at the North Wales Hospital for Nervous and Mental Diseases.

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Surgery: A Concise Guide to Clinical Practice. Edited by GEORGE L. NARDI, M.D., and GEORGE D. ZUIDEMA, M.D. 9¼ × 6¼ in. Pp. 1034 + viii, with 130 illustrations. 1961. London: J. & A. Churchill Ltd. 60s.

THIS surgical guide is composed of some forty-five chapters contributed by twenty-three authors. Every aspect of clinical surgery is covered, including biopsy principles, radio-active isotopes, cancer chemotherapy, and industrial hazards and emergencies. A whole chapter is devoted to laboratory tests which are useful in modern surgical diagnosis. A chapter on diet tables is also included.

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Development and Structure of the Cardiovascular System. Edited by ALDO A. LUISADA, M.D. 10 × 7 in. Pp. 148, with 100 illustrations. 1961. London: McGraw-Hill Publishing Company Ltd. 77s.

THIS is an interesting monograph sponsored by the American College of Cardiology. It is made up of a series of articles by eleven contributors, one of whom is Professor G. A. G. Mitchell of Manchester.

There is an excellent account of the embryology of the heart and great vessels, and the drawings illustrating these developmental processes are pre-eminent. The anatomy of the heart and its vessels is well described and presented. Professor Mitchell writes on the innervation of the heart and vessels and he has managed to put forward in a very lucid way the various connexions of the sympathetic outflows. He has included some very original drawings depicting the main nerves of the heart and their cerebral connexions. It is very stimulating to see new and original anatomical drawings in these modern times.

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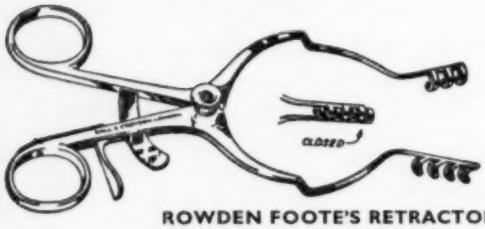

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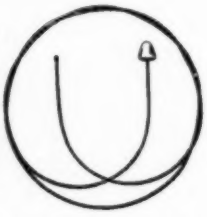

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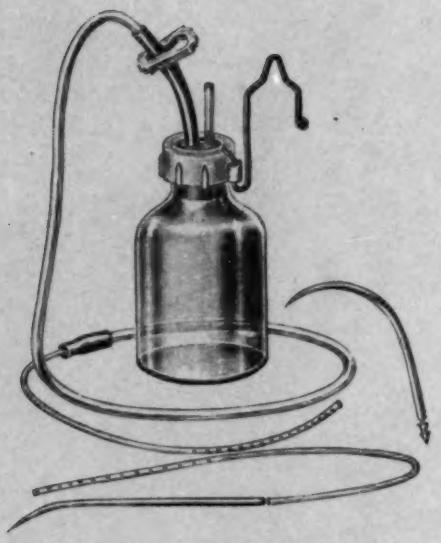

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